COMPARISON OF CLINICO-BACTERIOLOGICAL PROFILE OF PYODERMA IN CHILDREN & ADULTS

A THESIS SUBMITTED TO THE BABA FARID UNIVERSITY OF

HEALTH SCIENCES, FARIDKOT (PUNJAB)

FOR THE AWARD OF DEGREE OF

DOCTOR OF MEDICINE

IN

DERMATOLOGY, VENEREOLOGY & LEPROSY

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Dedicated

to

my teachers

family

&

friends

ACKNOWLEDGEMENT

As I pen down this thesis, overwhelming gratitude and reverence propel me to acknowledge the mighty and hands that have shaped up this work.

The instinctive qualities and utmost skills of my respected guide have paddled me through this challenging journey. **Dr. Sunil K Gupta**, Professor and Head, Department of Dermatology, Dayanand Medical College and Hospital, Ludhiana. Sir, kindly accept my most humble and heartfelt gratitude for investing your unmatched expertise, wisdom and enormous consideration throughout this work. I feel blessed under your guidance. He is the epitome of understanding, accessibility and helpfulness, who has provided me with monumental support. I am grateful to him for his intellectual inspiration and encouraging attitude and teaching me the fundamentals of a good clinician, dedication, patient care, and unquenchable thirst for acquiring knowledge and newer skills in dermatology. He is the “light of inspiration” I will always follow! Working under his guidance has been indeed a matter of pride for me.

My pen blooms with energy as I extend my utmost gratitude to my co-supervisor, **Dr. Veenu Gupta**, Professor, Department of Microbiology, Dayanand Medical College, Ludhiana. Your relentless support, encouragement and cosmic kindness have set the right framework for me to keep going. I am humbled by your positivity.

As I introspect the ground under my feet, I feel submerged with obligation towards **Dr. Sukhjot Kaur**, Associate Professor, Department of Dermatology, Dayanand Medical College, Ludhiana, my co- supervisor. I feel short of words to acknowledge her unconditional support through thick and thin. With a personality of masterly finesse, she keeps radiating knowledge and inspiration.

I extend my sincere thanks to **Dr. Jaspriya Sandhu**, Assistant Professor, Department of Dermatology, Dayanand Medical College,

Ludhiana for her invaluable support and guidance, which essentially aided this dissertation. She is a major pillar of the foundation that sustains me on a foreign land.

I also express my gratitude to my seniors and colleagues Dr. Navneet, Dr Arushi, Dr.Sukhleen, Dr. Monika, Dr. Manisha, Dr. Shriya, Dr. Amandeep, Dr.Harleen and Dr.Jaismine with whom I had the pleasure to work with and without whom this task wouldn't have been possible.

I am also thankful to Mrs. Lathika, Mrs. Samila, Mrs. Navdeep Kaur, Mrs. Kamlesh, Mr. Rai Singh, Mr. Jaipal, Mrs. Jagwati and Mr. Avtar Singh for their constant support and love during this journey to my dreams. My special thanks to the entire staff of Skin and R&D department DMCH, for their help in the accomplishment of this dissertation.

Some invisible forces akin to the Supreme force have motored me throughout. I am mesmerized by the amount of support I could ever get from my family.

An ultimate tribute goes to all my patients.

Dr. Aditi Bansal

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ABBREVIATIONS

A. hydrophila	*Aeromonas hydrophila*
AIDS	Acquired Immuno Deficiency Syndrome
ASO	Anti-streptolysin O
B. anthracis	*Bacillus anthracis*
B. brevis	*Brevibacillus brevis*
B. burgdorferi	*Borrelia burgdorferi*
B. henselae	Bartonella henselae
BHS	β-hemolytic streptococci
BV	Bacterial vaginosis
C. difficile	*Clostridium difficile*
C. diphtheriae	*Corynebacterium diphtheriae*
C. minutissimum	*Corynebacterium minutissimum*
C. trachomatis	*Chlamydia trachomatis*
C. perfringens	*Clostridium perfringens*
CA-MRSA	Community-acquired Methicillin-resistant S. aureus
DNA	Deoxyribonucleic Acid
E. coli	*Escherichia coli*
E. rhusiopathiae	*Erysipelothrix rhusiopathiae*
FDA	Food and Drug Administration
H. influenzae	*Haemophilus influenzae*
HA-MRSA	Hospital-acquired Methicillin-resistant *S. aureus*
HIV	Human Immunodeficiency Virus
K. pneumonia	*Klebsiella pneumoniae*
M. leprae	*Mycobacterium leprae*
M. morganii	*Morganella morganii*
MRSA	Methicillin-resistant *Staphylococcus aureus*
MSSA	Methicillin-sensitive *Staphylococcus aureus*
N. gonorrhea	*Neisseria gonorrhoeae*
N. meningitidis	*Neisseria meningitidis*
P. acne	*Propionibacterium acnes*
P. aeruginosa	*Pseudomonas auroginosa*
P. fluorescens	*Pseudomonas fluorescens*
P. mirabilis	*Proteus mirabilis*
P. ovale	*Pityrosporum ovale*
P. putida	*Pseudomonas putida*
PABA	p-aminobenzoic acid
PBP	Penicillin-binding proteins
PVL	Panton-Valentine leucocidin

RNA	Ribonucleic Acid
S. agalactiae	*Streptococcus agalactiae*
S. aureus	*Staphylococcus aureus*
S. epidermidis	*Staphylococcus epidermidis*
S. maltophilia	*Stenotrophomonas maltophilia*
S. marcescens	*Serratia marcescens*
S. pneumoniae	*Streptococcus pneumoniae*
S. pyogenes	*Streptococcus pyogenes*
S. venezuelae	*Streptomyces venezuelae*
SSTI	Skin or soft tissue infections
TMP-Sulfa	Trimethoprim-Sulfamethoxazole
VISA	Vancomycin-intermediate *S. aureus*
VRE	Vancomycin-resistant *Enterococcus*
VRSA	Vancomycin-resistant *Staphylococcus aureus*
Y. pestis	*Yersinia pestis*

ABSTRACT

Introduction: Pyogenic infections are characterized by local and systemic inflammation, formation of pus, generally caused by one of the pyogenic bacteria. They constitute a considerable proportion of cases worldwide and 17% of cases in India. The spectrum of organisms that causes pyodermas changes over time and have also developed multi drug resistance thus, the emergence of antibiotic resistant pathogenic bacteria is considered as grave threats to the public health worldwide.

Aim of study: To study the clinical manifestations and bacteriological profile of pyoderma in children and adults.

Materials and methods: A total of two hundred (divided into two groups of 100 each, first group <18 years and second group >18 years) clinically diagnosed cases of pyodermas seen in the outpatient and inpatient department of Dermatology of DMCH, Ludhiana were included in this study. Detailed history of the patients was taken, pus samples were collected and processed in the Department of Microbiology. Gram staining and Culture and sensitivity of all the samples was done.

Results: Among clinical diagnosis of primary pyodermas (72%), impetigo contagiosa was the most common clinical presentation (63%) in children while folliculitis (29%) was seen in adults. Secondary pyodermas were observed in 28% patients, from which infected scabies (47%) ranked the highest in children while infected tinea (57%) in adults. Pus culture confirmation was done. *S. aureus* was the most common isolate in both children (92%) and adults (65%) among gram positive organisms whereas among gram negative organisms, *E. coli* (4%) was cultured in children whereas *P. aeruginosa* (14%) in adults. Diabetes and poor hygiene were significantly associated with pyodermas (p value-0.00).

Conclusions: Pyodermas are common skin problem often because of therapeutic failure due to resistance of organism to some of the antibiotics leading to difficulty in treatment. Hence, the study of clinical diagnosis of pyodermas and correlation of bacteriological profile with antibiotic susceptibility can be helpful in tackling it.

Keywords: Primary pyoderma, Secondary pyoderma, MRSA, Impetigo contagiosa, Furuncle, Folliculitis, Antibiotic susceptibility.

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INTRODUCTION

Pyoderma is one of the commonest conditions encountered in dermatological practice. Studies indicate that up to 17% of the clinical visits may be for bacterial skin infections.[1] Studies indicate that among skin diseases affecting school children, incidence of bacterial pyoderma (impetigo, folliculitis and infected bite reactions) was 64.4% among all skin infections.[2] The preponderance in the preschool age group is due to the fact that these children are more exposed to unhygienic conditions when they are left under improper supervision by the parents who are working during the day.

Pyodermas can be primary or secondary which is superimposed on other dermatoses or traumatic or post-surgical wounds. Primary pyoderma is a pyogenic infection of the skin (non-diseased) and its appendages. It includes impetigo, folliculitis, furunculosis, carbuncle and ecthyma.

The secondary pyoderma is a pyogenic infection of previously diseased skin and it includes infected eczema, infected scabies, infected wounds and trophic ulcers etc.

Pyodermas can be classified as 'superficial' (epidermis and dermis) and 'deep' (hypodermis, fascia and muscle).[3] It can be community acquired (CA) or hospital acquired/ nosocomial (HA) infection. The former usually involves a single pathogen, whereas the latter is often polymicrobial.

Though easily treatable, it is known for its chronicity, recurrence, and other complications. Recurrent pyoderma is a problematic infection experienced by children as well as adults with or without underlying dermatological disorders or immunodeficient states. Recurrence may result from the following causes:

- Inadequate treatment or inappropriate choice of antibiotics

- Underlying skin disease which was left uncared for during treatment of infections, e.g., scabies, atopic dermatitis, tinea capitis, contact dermatitis and psoriasis.
- Colonization with drug-resistant strains of bacteria; there is an increased incidence of skin colonization with CA-MRSA in children.[3]
- Underlying immunosuppression

Various predisposing factors for developing cutaneous infections include poverty, malnutrition, overcrowding, illiteracy, customs, habits, patients on treatment with steroids or chemotherapeutic agents and those with pre-existing skin diseases (scabies, atopic dermatitis, tinea capitis, contact dermatitis and psoriasis), obesity , immunodeficiency, diabetes, chronic granulomatous disease, defects in the mucocutaneous barriers produced by trauma including surgery, foreign surfaces (sutures, shunts, intravascular catheters) and burns.[4]

Climatic conditions also play a role, with the hot and rainy seasons being the period of maximum occurrence.[5] The incidence of pyoderma cases is increased during the hot and rainy seasons as conditions during this period including microtrauma caused by biting insects predispose the susceptible patient to these infections.

Immunocompromised states related to primary immunodeficiency disorders or those secondary to malignant diseases or immunosuppressive therapy pose special problem in the management of pyodermas in children. Neutropenia is common in children on cancer chemotherapy, and such children are at special risk of developing infection (cellulitis/ ecthyma gangrenosum) by Gram-negative organisms like *Pseudomonas aeruginosa.*[6] HIV-infected children are at higher risk of developing both CA-MRSA and HA-MRSA infections, and are associated with increased mortality.[4]

Infection is usually polymicrobial in immunocompromised children, common organisms being *S. aureus* and *P. aeruginosa,* which require broad-spectrum antimicrobial coverage.

Children with risk factors for developing HA-MRSA infection are more likely to develop invasive disease.[7] A higher prevalence of pyodermas in children is of significance as they are most susceptible to non - suppurative complications like glomerulonephritis and Rheumatic fever.

Skin infections are more commonly seen in infants and children than adults. This is because of the difference in infants and children's skin as compared to adult's skin. Although the structure is completely same, but there is a difference in maturity and function. Infant's and toddler's skin is thinner as compared to adults skin. Both natural and adaptive body's defense system in the skin is not mature enough, causing immature protective function against infections in infants and children.

Pyoderma is said to be complicated- when deeper structures like fascia and muscle are involved, necessitating surgical intervention, when the patient has associated co-morbidity like diabetes mellitus or other immunosuppressed states, affecting the response to usual treatment, when they involve the perineal and/ or perianal region, with the risk of infection by anaerobic and Gram-negative pathogens.[3]

Staphylococcus and *Corynebacterium* spp. are the most abundant organisms colonizing moist areas. These moist sites include the umbilicus (navel), the axillary vault, the inguinal crease (side of the groin), the gluteal crease (topmost part of the fold between the buttocks), the sole of the foot, the popliteal fossa (behind the knee) and the antecubital fossa (inner elbow).[8]

The most diverse skin sites are the dry areas, with mixed representation from the phyla Actinobacteria, Proteobacteria, Firmicutes and

Bacteroides.[8] These sites include the forearm, buttock and various parts of the hand.

Most of the pyodermas are caused by mixed bacterial flora. Aerobic and anaerobic, gram-negative and gram-positive organisms, whose origins are from the oral, gastrointestinal and skin flora may be present in such infections, where they exist synergistically.

The common gram-positive organisms responsible for pyodermas are *Staphylococcus aureus* and *Streptococcus pyogenes*. The gram-negative organisms are *Escherichia coli, Klebsiella* species*, Pseudomonas aeruginosa, Enterobacter* species*, and Proteus* species.[8]

The majority of the primary and secondary pyodermas are caused by either *S. aureus* or group A *Streptococcus*. These bacteria cause a broad spectrum of infections, ranging from superficial pyodermas to invasive soft tissue infections.[9]

S. aureus is the commonest pathogen isolated from pyodermas in more than 70% of all cases of pyodermas[6] and in approximately 50% of cases of cellulitis.[10] In a study of 250 cases of pyoderma at all ages, *Staphylococcus aureus* was isolated in 80.8% of cases.[11] *Staphylococcus aureus* is responsible for over 70% of all skin and soft tissue infections in children and accounts for up to one-fifth of all visits to pediatric clinics.[4]

Methicillin-resistant *Staphylococcus aureus* (MRSA) was recognized initially in the health-care setup (1960s), followed by its spread in the community (1980s)[12] MRSA is defined as isolates with a methicillin minimum inhibitory concentration ≥4 μg/mL. Methicillin resistance to *S. aureus* is mediated by mecA gene which encodes a novel altered penicillin-binding protein-2A which induces resistance to all β-lactam antibiotics including cephalosporins.[13] The incidence of MRSA is variable

(1%-74%) in the geographic areas and among various communities in the different countries.[12]

Currently, community-acquired MRSA (CA-MRSA) has emerged as the most commonly identified pathogen in cases of pyodermas in many areas and accounts for >90% of bacterial skin infections in children. Outbreaks of CA-MRSA SSTIs are prevalent in child care centers and newborn nurseries.[3] Some CA-MRSAs have Panton-Valentine-Leukocidin (PVL) genotype, which contributes to severe, necrotizing skin infections.

The possible clinical indicators of MRSA infections are as follows:

1. Evidence of concomitant lung (associated cough) and skin infection
2. Presence of localized peri-umbilical folliculitis
3. Excessive pain, beyond clinician's expectation

The spectrum of cutaneous bacterial disease is forever changing. Increasing resistance to antibiotics seen in micro-organisms poses a big problem to the clinicians. Therefore, timely recognition and prompt bacterial diagnosis with antimicrobial sensitivity is imperative for the effective management and treatment of pyoderma.

The emergence of antibiotic resistance has significantly deteriorated the utility of established antibiotics and it poses a serious threat to the public health .Choice of antimicrobial agents should ideally be guided by bacterial culture and sensitivity testing from the infective material collected from the site of infection (e.g., pus, aspirated fluid). In children, it may be difficult to perform invasive procedures like aspiration, and in such situations surface swab from the infected site may be taken.

Swabs of skin lesions should be taken in all types of skin infections, not only to identify the organisms responsible for the infection but importantly, to determine their anti-microbial sensitivities in case empiric first-line treatment fails.

Management

The management of skin infections requires careful history taking and clinical examination. In children with bites or puncture wounds, careful assessment of the risk of tetanus, rabies, and other bite-related infections needs to be made. Particular attention should be paid to the presence of any systemic symptoms and signs, such as fever and rigors, which may indicate bacteremia and possible secondary bacterial infection including pneumonia and meningitis.

Children who are immunocompromised for various reasons – such as primary immunodeficiency diseases, malignancies, chemotherapy, AIDS, and on long-term corticosteroid therapy – need careful monitoring, because they may deteriorate rapidly.

Majority of the superficial bacterial infections can be managed on an outpatient basis (outpatient management)**.** The decision of inpatient management (hospitalized management) depends on the following factors-[4]

- Depth and extent of the infection
- Unusual clinical presentation
- Presence of complications/ risk factors
- Systemic involvement
- Underlying immunosuppressed state

Occasionally, superficial infections like erysipelas and cellulitis may necessitate hospitalization if signs of rapid progression develop.

Many cases do not respond to some antibiotics which were previously very effective for such cases, owing perhaps to the indiscriminate use of topical and systemic antibiotics.

Since culture and sensitivity results are available approximately after 72 hours, empirical antibacterial therapy can be started, followed by streamlining of antibiotic therapy once the reports are in hand.

A few localized lesions without systemic symptoms (fever, lymphadenopathy) or other signs of systemic infection (raised white cell count and inflammatory markers) can be treated with topical antibiotics such as - mupirocin or topical fusidic acid.

Multiple or more extensive infection in an otherwise well child should be treated with oral antibiotics and assessed at 24–48 hours. If there is no improvement or the lesions have progressed significantly, then intravenous antibacterial therapy is warranted.[4]

Some patients may require prolonged low-dose oral antibiotics to prevent recurrent infections.

Many of the newer classes of anti-microbials are not freely available in different parts of India and even if available are not affordable by people from low socioeconomic strata, who are the common sufferers of skin infections.

Many cases these days do not respond to antibiotics and this may be attributed to indiscriminate use of topical and systemic antibiotics. Hence, timely recognition, and prompt bacterial diagnosis of the cases is very important for the management and also to check the major complications.

Therefore, pus culture and sensitivity tests in pyoderma are highly recommended to identity the commonly prevalent pathogens and to aid in the judicious use of cost - effective antibiotic & to prevent bacterial resistance.

Though the problem of drug resistance exists, while treating skin infections in India, the clinicians in primary health-care setup do not have many options except empirical antibiotic treatment available, cheaper drugs like - penicillin’s, co-trimoxazole, erythromycin, topical gentian violet etc. However, whenever, no response to therapy is suspected, the option of referral to higher centers should be kept open.[3]

In secondary and tertiary health-care centers, there must be an attempt to identify prevalent organisms causing bacterial skin infections in the locality and the emerging antibiotic-resistance pattern.

In this regard, periodic collaborative works among all clinical departments, along with collaboration with microbiologists, is helpful. This will help to chalk out effective management protocol, keeping in mind the limited resources, for bacterial skin infections in developing countries.

AIMS AND OBJECTIVES

1. To study the clinical manifestations of pyoderma in children and adults.
2. To study their etiological agents and correlate clinical and bacteriological findings.

REVIEW OF LITERATURE

Pyoderma has been known since ancient time, but etiology was unknown for a long period of time. In 1546, Fracastorius of Veruna proposed a contagium, vivum as the possible cause of infectious diseases.[13]

As the bacteria are not visible to naked eye, their role in the etiopathogenesis of diseases was unknown till the discovery of microscope in 1683 by Anton Von Leeuwonhoek. He first observed and described bacteria.

In 1871, *Staphylococci* were first observed in human pyogenic lesions by Von Recklinghausen. Sir Alexander Ogsten, gave it the name because of its typical appearance of cocci in grape like clusters.[13]

In 1884, Rosenbach suggested the name *Staphylococcus aureus* for pathogenic strains producing golden-yellow colonies and term *S. alba* for the strains from normal skin which produce white colonies on culture medium.

Pyodermas can be endogenous or exogenous. Any abrasion in the skin provides an entryway to the surface bacteria, and these bacteria stick to the edges of the abrasion. They are characterized by pus formation which is caused by pyogenic bacteria, which accumulate dead leukocytes in the form of pus.[14] Coagulase positive *S. aureus* has been found to be more dominant organism in pus followed by Streptococcus spp.[15,16]

Various factors like poverty, malnutrition, overcrowding and poor hygiene are responsible for its higher incidence in the lower socio-economic strata.[5] Climatic conditions also play a role with the hot and rainy seasons being the period of maximum occurrence.

Bacterial skin infections are the commonest infections in Indian pediatric population and in other developing countries and contribute to significant morbidity and increased mortality.

Parents often neglect to seek health-care facilities for superficial pyodermas, because of the trivial nature of the lesions and can lead to crippling complications like chronic glomerulonephritis or rheumatic heart disease in adult life. Hence, general population, like groups of women, school teachers and primary health-care workers should be made aware about the importance of early diagnosis and treatment of bacterial skin infections.

Normal flora of skin:

Microbial colonization of the skin provides defense against the potentially pathogenic organisms. Normally, microbes live in synergy with their hosts but, occasionally colonization can result in clinical infection.[17]

Normal skin microflora can be classified into three groups: -

1. **Resident flora**-These organisms grow on the skin and are stable in number and composition.

2. **Transient flora**-These organisms lie free on the skin surface without attachment and are derived from exogenous sources. This group varied widely in number and type and are found primarily on exposed skin.

3. **Nomads or temporary residents**- These organisms are called so because of their tendency to adhere to and multiply on the skin for only a short period. They are also known as associate flora because they are often dependent for their proliferation on the activities of the resident flora and environmental influences. They colonize the skin in minority of persons.

Resident skin flora-

1. Micrococcaceae- Coagulase-negative staphylococci *Peptococcus* *Micrococcus* species
2. Coryneform organisms- *Corynebacteria* *Brevibacterium*
3. *Propionibacterium*
4. *Acinetobacter*
5. *Pityrosporum*

Flora of specialized areas-

Flora from any one area of the skin cannot be taken as representative of the entire flora. Certain parts of the skin have specific resident flora which differ in quantity as well as the quality from the general normal flora of integument.

1. Nasal Vestibule-

In 25% of healthy individuals, coagulase negative staphylococci, *Staphylococcus aureus* and *Micrococci* are present as normal inhabitant of nasal vestibule.

2. External auditory meatus-

Coagulase negative staphylococci, *Coryneform*, *Proteus* species, *Escherichia coli* and *Neisseria catarrhalis* constitute normal flora of external auditory canal.

3. Moist areas

a. **Axillae**:

Some individuals may have *Staphylococci*, whereas in some *Propionibacterium acnes* may predominate.

b. **Toe cleft**:

The toe cleft is often colonized by a large number of species such as- *Brevibacterium, Acinetobacter, Coliform*. The colonization of the cleft is because of occlusion, hyperhidrosis and maceration. It is often exacerbated by wearing tight footwear.

c. **Vulva, perineum and groin**:

Coagulase negative staphylococci, *Micrococci*, *Coliform enterococci*, group B Streptococci and *Acinetobacter* species constitute resident and transient flora in these sites.

4. Umbilicus:

The umbilicus of the newborn is frequently colonized by *S. aureus* shortly after birth. It may also be colonized by *Streptococcus pyogenes* which may be responsible for nosocomial infections in hospital nursery and maternity wards.

5. Hair follicles:

Propionibacterium acnes is found in the deeper parts of hair follicle whereas aerobic cocci and *P. ovale* colonize near to the surface.

ROLE OF NORMAL FLORA [17]

a) They provide defense mechanism against bacterial infection through bacterial interference.

b) They help in antibody production.

c) They produce free fatty acids by hydrolyzing skin lipids mainly sebum, which provides defense against bacteria.

d) They are responsible for odor production in axilla by their action on apocrine sweat.

Classification of Pyoderma-

Pyodermas may be mild or severe.

Primary pyodermas:

They are pyogenic infection of non-diseased skin and its appendages.

A. Impetigo

1. Impetigo bullosa/ bullous impetigo
2. Impetigo contagiosa/ non bullous impetigo

B. Folliculitis

1. Superficial:

a) Follicular impetigo/ Bockhart's impetigo

b) Hot tub folliculitis

2. Deep:

a) Sycosis barbe

b) Pyoderma faciale

c) Folliculitis decalvans

C. Furuncle

D. Carbuncle

E. Paronychia

F. Ecthyma

G. Erysipelas

H. Cellulitis

I. Erythrasma

Secondary pyodermas:

They develop in areas of already damaged skin, in contrast to primary infections. They often show a mixture of organisms on culture.

1. Burns

2. Eczematous dermatitis
3. Chronic ulcers
4. Dermatophytosis
5. Traumatic lesions
6. Vesicular/ bullous eruptions
7. Scabies

Primary Pyoderma-

1. **Impetigo**:

It is a common cutaneous infection that is especially prevalent in children. It was described by Tilbury Fox in 1864.[18]

Traditionally, it was caused by either group A β-hemolytic *streptococci* or *Staphylococcus aureus*. Currently, it is most commonly caused by *S. aureus*.

Engman in 1901, distinguished between Streptococcal and Staphylococcal impetigo clinically, Streptococci was responsible for thicker crusts as compared to *Staphylococci*.

It is of 2 types-

a. Impetigo contagiosa, also known as non-bullous impetigo
b. Bullous impetigo.

a. **Non-bullous impetigo, or impetigo contagiosa** :

It is caused by *S. aureus* or *S. pyogenes*, and is characterized by honey-colored crusting mainly on the face and extremities. It is common in adults and children but rarely in those under two years of age.

Crusted impetigo can occur in normal skin or over pre-existing dermatosis such as -atopic dermatitis, contact dermatitis, insect bites, pediculosis and scabies. The main predisposing factors are malnutrition and poor hygiene.

The initial lesion is a vesicle, 1 to 2 cm in diameter, located on an erythematous base, which is easily ruptured, causing superficial ulceration which is covered with honey-colored crusting. Satellite lesions, which are

due to self-inoculation are quite common. There is predominance of lesions over exposed areas, especially on the limbs and face. Regional lymphadenopathy is common and fever can occur in severe cases. It may resolve spontaneously without any treatment in 2-3 weeks.[19]

In 2007, retapamulin was the first agent for human use approved in the pleuromutilin class of antibacterial in the United States (U.S.), and is the first topical antibacterial indicated to treat impetigo in over 20 years.[20]

b. **Bullous impetigo**: It is caused by *S. aureus,* phage type 71 (60% of cases). Other phage types involved are 3A, 3C and 55.

It presents with large, flaccid bullae up to 2 cm in diameter and is commonly seen after the second week of life, although it can be present at birth in case of premature rupture of membranes. It is most commonly seen among children aged two to five years.

Initially the bulla has clear content that later becomes purulent. The roof of the blister ruptures easily, which reveals an erythematous, shiny and wet base. It involves intertriginous areas such as diaper area, axillae and neck, although any cutaneous area can be affected, including palms and soles. Lymphadenopathy is usually not seen.

S. aureus produces exfoliative toxins, which are the proteases that selectively hydrolyze desmoglein-1, which is an intracellular adhesion molecule present in the desmosomes of keratinocytes located in the granular layer of epidermis. Toxins are the greatest virulence factor of *S. aureus*, causing dissociation of epidermal cells with formation of blister. There are two different types of exfoliative toxins, namely exfoliative toxin A related to bullous impetigo and toxin B with scalded skin syndrome.

It is usually a self-limited condition. Rarely, complications can occur which include- cellulitis, septicemia, osteomyelitis, septic arthritis, lymphangitis, lymphadenitis, guttate psoriasis, staphylococcal scalded skin syndrome

and the most serious complication, post-streptococcal glomerulonephritis.[21]

2. **Folliculitis**: It is a follicular based pustule due to infiltration of the cells within the follicular wall and ostium.

In superficial folliculitis, the inflammation is restricted to the infundibulum of the follicle, whereas in deep folliculitis the inflammation not only involves the deeper aspect of the follicle, it also extends into the surrounding dermis and can result in scarring.[22]

In perifolliculitis, there is infiltration of lymphocytes within the perifollicular tissues and it also extends into the adjacent reticular dermis.

Inflammation of hair follicles can occur after physical injury, chemical irritation, or infection.

Superficial folliculitis was *described by Bockhart in 1887.It* is caused by *S. aureus*, as the result of inflammatory changes confined to the follicle orifice and is also known as follicular impetigo or Bockhart impetigo. It is commonly seen on the scalp and limbs, but can also be seen on the face, especially periorally. The lesions are small, discrete pin-head sized, fragile, yellowish-white, dome shaped pustules over an erythematous base and are located at the ostium of the pilosebaceous canals associated with mild itching or burning. It develops in crops and heals in a few days without scar formation.

It is associated with poor hygiene, maceration and drainage from wounds and abscesses.

Chronic folliculitis of the legs predominantly affects those in age group of 15-30 years. It is commonly seen in Asians and Africans, especially young adult males in India. In this, the inflammatory changes are confined to the ostium or slightly below it and heals with atrophic scarring. Apart from coagulase positive *S. aureus*, physical or chemical injury may also precipitate this condition. It involves the legs, thighs, forearms and arms.

Topical and systemic antibiotics and avoidance of risk factors are essential in controlling the disease.

a) *S. aureus* folliculitis	Superficial (follicular/Bockhart's impetigo) Deep (sycosis)
b) *P. aeruginosa* folliculitis	Hot tub folliculitis
c) Gram negative folliculitis	*Klebsiella, E. coli, Proteus*

3. **Sycosis barbae**: It was described by Thomas Bateman in 1814.[13] It is presented as severe, painful, deep, recurrent form of facial folliculitis (young black males are particularly affected) and is characterized by inflammation that involves the entire depth of the hair follicle.

Folliculitis barbae, a perifollicular chronic staphylococcal infection of the beard area, is defined as persistent irritation caused by shaving and commonly occurs in men aged 20 to 40 years. It is most frequently seen as superficial pustules pierced by hairs on an erythematous base that can be asymptomatic or painful and tender. If untreated, the infection can gradually progress leading to a deep-seated infection known as sycosis barbae and results in atrophic scarring.[22]

4. **Furuncle**: It is characterized by deeper accumulation of pus and necrotic tissue within the hair follicle. They appear on the hair-bearing areas of the skin and most commonly caused by *S. aureus*.

Clinically, it presents as erythematous, edematous and tender nodules of varying sizes. Fever and enlarged lymph nodes are rarely seen. It is mainly seen on the extremities and may heal with scarring.

It has a propensity to recur and, in such cases, they often spread among family members. Obesity, malnutrition, diabetes, immunosuppression, hyperhidrosis, dermatitis, maceration and friction predispose to the development of furuncles.

Recurrent furunculosis is defined as three or more attacks within a period of 12 months. Colonization of the anterior nares by *S. aureus* plays a definite role in the etiology of chronic or recurrent furunculosis. Besides the nares, colonization is also seen behind ears, under pendulous breasts, and in the groins. Enteric bacteria are responsible for furuncles occurring in the perineal area. *Corynebacterium*, *S. epidermidis*, and *S. pyogenes* may also be seen in furunculosis. Immunodeficiency is rarely the primary cause.

Recurrent furunculosis is mainly caused by *MSSA.* Now-a-days, CA-MRSA has gained popularity as the most common cause of pyodermas occurring in the emergency room in most of the countries.

Some strains of MRSA, particularly the CA-MRSA, produces a toxin named Panton-Valentine leukocidin (PVL) and are associated with severe infections of skin. PVL is leukocidal, and severe but rare complications such as necrotizing fasciitis and necrotizing pneumonia are described following soft tissue infection with MRSA. PVL is a virulence factor of *S. aureus* which correlates with chronic recurrent furunculosis.[23]

Scarring and recurrence are the most common complication. Added complications include-pneumonia, necrotizing fasciitis, myositis, osteomyelitis, septic arthritis and meningitis, which are rarely seen.

5. **Carbuncle**: It is also known as infective gangrene of the skin and subcutaneous tissue, which usually starts as a furuncle around the root of the hair follicle and *S. aureus* is mostly isolated. It is infection of a group of adjoining follicles that form a large, swollen, tender masses which have multiple draining points with surrounding inflammation. It may be

accompanied with fever, leukocytosis, and bacteremia. In uncontrolled diabetes or other immunocompromised states, the infection may spread in the subcutaneous plane and burrow upwards forming multiple draining pus points or sinuses. The diagnosis is frequently delayed as it occurs on the back or nape of neck, hence, not easily visualized. Carbuncles are not uncommon even in this post antibiotic era especially in those with uncontrolled diabetes. Early diagnosis and surgical management are essential.

Large or deep nodular lesions may require incision and drainage. Lesions usually heal with scarring.

6. **Cellulitis**: Any spreading infection involving the dermis and subcutaneous tissues. Purulent cellulitis is defined as cellulitis with associated purulent exudate in the absence of a drainable abscess.[24]

Purulent cases are more likely to be caused by *S. aureus* including CA-MRSA. Empiric antimicrobial therapy in these cases should be directed against CA-MRSA, and is modified based on cultures. More commonly, cellulitis is non-purulent. Antimicrobial therapy in such cases should be directed at β -hemolytic streptococci and methicillin-sensitive *S. aureus*. Animal bites are associated with cellulitis due to Gram-negative organisms such as *Pasteurella* and *Capnocytophaga*. Exposure of abraded skin to salt or fresh water is associated with Vibrio vulnificus and Aeromonas spp. respectively.[25]

Group A streptococci can be associated with the development of necrotizing fasciitis, although this can also be due to mixed infection including Gram-negative and anaerobic organisms, particularly in the elderly and immunosuppressed.

Patients with cellulitis typically present with a brief history of pain, redness, and swelling of the involved skin .Predisposing factors are obesity, prior saphenous vein removal, history of radiation therapy, or any skin disorder

such as-ulcers, wounds, dry skin with fissures, chronic skin diseases, and venous stasis .Toe web intertrigo also has been shown to be a risk factor for cellulitis, especially if it is complicated by bacterial colonization with pathogenic bacteria.[26] The most common site of involvement in cellulitis is lower limbs followed by upper limbs, trunk, perineum and head and neck .On examination, it is warm, red, tender, swollen and may be associated with lymphangitis or lymphadenopathy. There may be associated vesicles and bullae. In erysipelas, the borders are well demarcated, whereas in cellulitis the borders are less well defined and bullae are filled with clear fluid as opposed to the hemorrhagic fluid which is found in necrotizing infections. It should be differentiated from eczema, lymphoedema and lipodermatosclerosis.[27]

7. **Erysipelas**: It involves more superficial dermal structures as compared to cellulitis and is distinguished clinically by raised borders and clear demarcation between involved and uninvolved skin. It is characterized by an inflammatory reaction of the upper dermis with a sharp demarcation of the erythema. Erysipelas is also referred to as “St. Anthony’s Fire” due to its intense fiery rash. Occasionally, a primary lesion i.e. wound or skin crack is present. It is associated with nausea, pain and fever. Surgical incisions, insect bites, stasis ulcerations, obesity, lymphedema, athlete’s foot, leg ulcers, eczema, intravenous drug abuse, poorly controlled diabetes, and liver disease and venous stasis are among the main entry portals. In addition, facial erysipelas could also be caused by a recent infection within the nasopharynx passage.[28] The distinction is significant because erysipelas is thought to be predominantly due to β-hemolytic streptococci (BHS). Traditionally, BHS are characterized by grouping and therefore the group isn't always in concordance with the species.[29,30]

The differential diagnosis includes -herpes zoster, contact dermatitis, urticaria, familial Mediterranean fever. It must be differentiated from

erysipeloid of Rosenbach, caused by *Erysipelothrix rhusiopathiae*, is usually unaccompanied by fever or any systemic symptoms.[30]

8. **Paronychia**: It is one of the most common infections of the hand. They are localized, superficial infections or abscesses of the perionychium and occur when there is an interruption occurs between the seal of the proximal nail fold and the nail plate which permits the entry of invading organisms.

Patients with acute paronychia complain of localized pain and tenderness of the nailfold. The nailfolds usually appears red and inflamed, and the nail plate may be discolored or even distorted. If left untreated, pus will accumulate beneath the nailfold which will extend beneath the nail margin to involve the nail bed, leading to elevation of the nail plate.[31]

Non-infectious causes of paronychia include contact irritants and excessive moisture. Paronychia can be acute or chronic. People with occupations like-baker, bartender and dishwasher are predisposed to develop chronic paronychia.

The most common infecting organism is *S. aureus*, followed by *streptococci, pseudomonas* and gram-negative organisms. Children are prone to acute paronychia secondary to finger sucking and nail biting.

Treatment includes warm-water soaks, antimicrobial therapy or surgical intervention. Acute paronychia is due to nail biting, finger sucking, aggressive manicuring, a hang nail or penetrating trauma, retained foreign body,[32] artificial nail placement.[33]

9. **Ecthyma**: It was first described in 1897 by Canadian pathologist Dr. Lewellys Barker as a manifestation of *Pseudomonas aeruginosa.*[34] Although *P. aeruginosa* remains the most frequent organism identified in EG, other causative pathogens are MRSA, *S.pyogenes*, *Citrobacter freundii*, *E. coli*, *A. hydrophila*, *K. pneumonia*, *S.*

marcescens, *S. maltophilia*, *M. morganii*, *C. diphtheriae*, *N. gonorrhea* and *Y. pestis*.

It most commonly occurs in immunocompromised individuals with fulminant bacteremia causing hematogenous spread of the bacteria.[35] It is also known to occur in otherwise healthy immunocompetent individuals.[36]

Risk factors include neutropenia, leukemia, multiple myeloma, diabetes mellitus, malnutrition, and extensive burn wounds.

It is mostly seen over gluteal or perineal area. Other sites of involvement are axilla, arms, legs, trunk, and face.

The initial skin lesion presents as a painless round erythematous macule, which rapidly turns into a pustule with surrounding erythema. In the center of the lesion a hemorrhagic bulla appears, which spreads peripherally evolving into a gangrenous ulcer with a central black or gray eschar surrounded by a characteristic erythematous halo, which is due to perivascular invasion and ischemic necrosis of the associated skin.

Early diagnosis and treatment with broad spectrum antipseudomonal agents are of critical importance. It denotes a poor prognosis, particularly in immunocompromised individuals with neutropenia. Multi-drug resistance to *P. aeruginosa* has increased in the recent years which makes treatment more challenging. Resistance mechanisms include decreased permeability (biofilm formation), antibiotic efflux pumps, antibiotic inactivating mechanisms, and antibiotic target site mutations.[37]

Secondary Pyodermas-

The skin lesions that are most commonly secondarily infected with bacteria are scabies,[38] psoriasis, [39] poison ivy, [40] atopic dermatitis, [41] eczema herpeticum, [42] and kerion.[43]

1. **Infected scabies**: It is also known as seven-year itch, and is caused by S*arcoptes scabiei var hominis*. The most common symptom is severe nocturnal itching.[44]

2. **Infected pemphigus**: It is a group of chronic autoimmune bullous dermatosis that are histologically characterized by the presence of bound and circulating autoantibodies directed against intracellular adhesions of epithelial cells.

3. **Infected contact dermatitis**: It is the inflammatory response of the skin to an exogenous substance which could be irritant or allergen.

4. **Trophic ulcer**: It is also known as phagedenic ulcer which is a result of synergistic bacterial infection, very common in hot and humid climate.

Causative Organisms:

There are various micro-organisms causing pyodermas. Most of them are described below:-

1. ***Staphylococcus aureus***: *S. aureus* is an aerobic, non-spore-forming, Gram-positive coccus belonging to the Micrococcaceae family, which is found in air, fomites, and dust, and commonly colonizes humans and animals.[45] It can be distinguished from other staphylococcal species on the basis of the yellow color pigmentation, β-hemolysis on blood agar, and positive results of coagulase, mannitol fermentation, and deoxyribonuclease tests. Higher carriage rates are seen in cases of atopic dermatitis, contact dermatitis, psoriasis, diabetes mellitus, AIDS, those undergoing hemodialysis, and surgery.[46]

S. aureus causes disease by local invasion and toxin production. The organism produces proteins and enzymes (protease, lipase, hyaluronidase etc.) that cause tissue destruction and spread of infection as well as protection against antibiotics (β-lactamases, penicillin-binding proteins, etc.). Like other skin commensals, usually it does not cause infection in healthy individuals.[47] However, when local barriers are broken, it causes skin infections and may invade into deeper tissues to cause serious systemic illnesses.

It is the most common cause of skin and soft tissue infections, including bullous and non-bullous impetigo, folliculitis, furunculosis, carbuncle, and cellulitis.[48] It is also responsible for surgical and traumatic wound infections, mastitis, and neonatal omphalitis.[49]

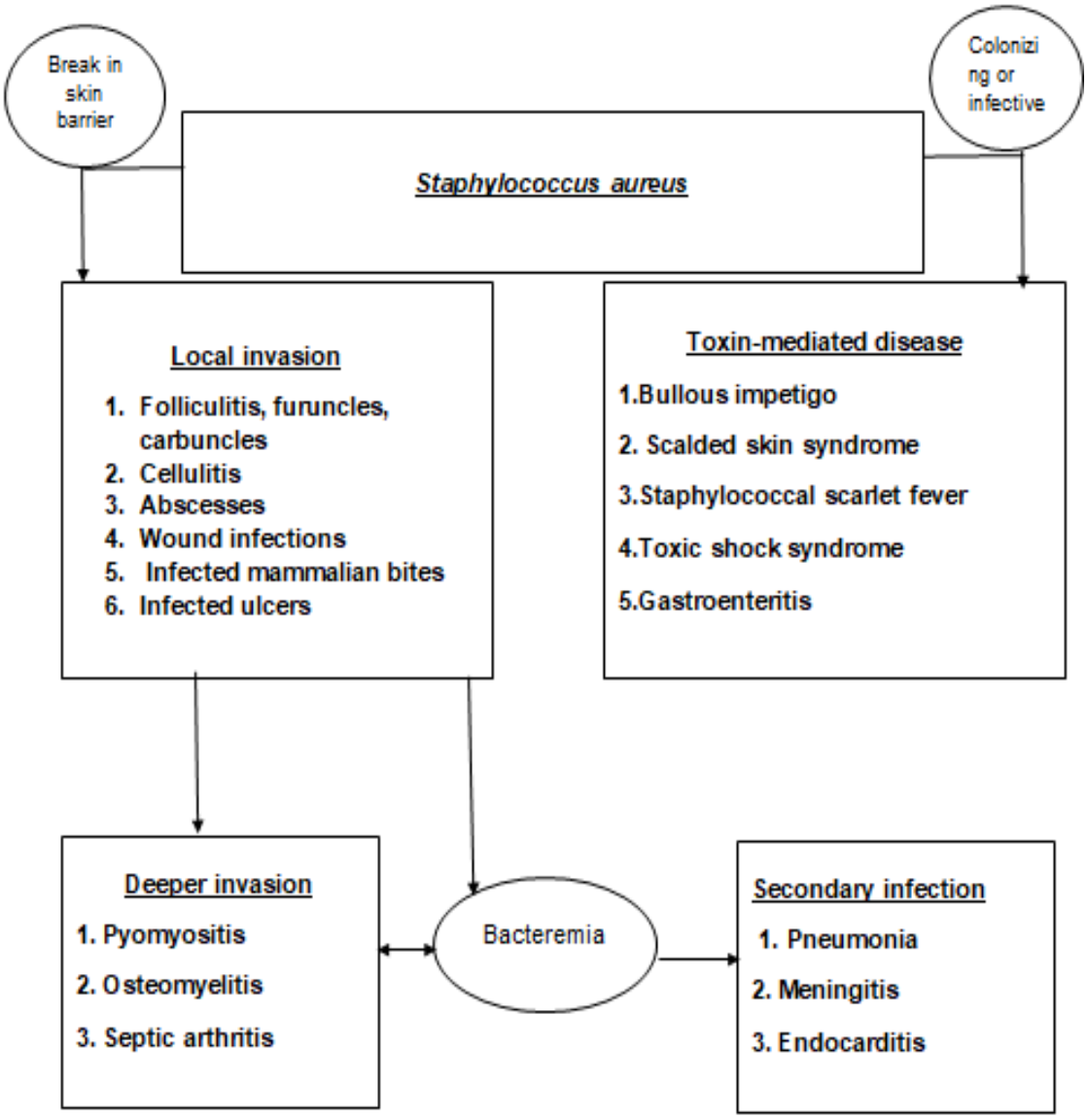
It is also able to cause disease by production of exotoxins such as enterotoxins, toxic shock syndrome toxin-1, and exfoliative toxins.

Like all other major pathogens, it is adapted to identify changes in its environmental conditions and regulate expression of different factors that alter colonization, multiplication, invasion, spread, destruction of host immune defenses, and development of multi-organ failure.[50] Its pathogenicity is multifactorial and depends on a wide range of cell wall-associated factors such as: capsular polysaccharide, Protein A, and fibronectin binding protein and extracellular virulence factors e.g. coagulase, hemolysins, enterotoxins, epidermolysins, and synergohymenotropic toxins produced by the organism.[51] The agr gene locus controls the expression of most of the virulence factors in *S. aureus* by increasing the production of extracellular proteins and suppressing the expression of cell surface proteins.[52-54] This locus encodes a two-component signaling pathway whose activating ligand is a bacterial density-sensing, auto-inducing octapeptide that is itself also encoded by the agr locus.

One particular virulence factor produced by *S. aureus*, called the PVL determinant, is strongly associated with primary skin infections .PVL, along with other leukocidins , belongs to a recently described family of synergohymenotropic toxins that damage host defense cells and erythrocytes by the action of two different secretory proteins.[55] PVL has not been detected in any HA-MRSA strains so far, but recently there are reports suggesting the association between PVL and CA-MRSA strains.[56] More than 90% of these strains were isolated from healthy children and young adults with skin and soft tissue infections.

There is also evidence that patients with HIV are at increased risk of *S. aureus* infections.[57] Several studies have reported *S. aureus* to be the most common pathogen responsible for bloodstream infections among HIV-positive patients in both the community and the hospital.

CA- MRSA strains are more likely to cause superficial skin and soft tissue infections. Risk factors include-prolonged or frequent hospital admissions, the presence of indwelling catheters, invasive or surgical procedures, critical care unit stay, prolonged or recurrent antibacterial exposure, endo-tracheal intubation, tracheostomy, chronic illnesses, and malignancy. Methicillin resistance is acquired through the mecA gene, which is carried in a larger genetic element called the staphylococcal cassette chromosome (SCCmec).



Infections caused by *Staphylococcus aureus* in humans

2. ***Streptococcus***: They are Gram-positive, nonmotile, non-spore forming, catalase-negative cocci that occur in pairs or chains. They lack the enzyme catalase. Older cultures may lose their Gram-positive character.

Mostly are facultative anaerobes, and few are obligate anaerobes. *S. pyogenes* is a round-to-ovoid coccus measuring 0.6-1.0 µm in diameter. It divides in one plane and thus occur in pairs, or in chains of varying

lengths. S *pneumoniae* is a 0.5-1.25 μm diplococcus, typically described as lancet-shaped. Group A streptococci have a hyaluronic acid capsule.

They are classified on the basis of colony morphology, hemolysis, biochemical reactions, and serological specificity. They are divided into three groups by the type of hemolysis on blood agar: α hemolytic (incomplete, green hemolysis), β-hemolytic (clear, complete lysis of red cells), and γ hemolytic (no hemolysis).

Serologic grouping is based on antigenic differences in cell wall carbohydrates (groups A to V), pili-associated protein, and in the polysaccharide capsule in group B streptococci. Some strains are nephritogenic.

Virulence factors of group A streptococci include-
(1) M protein and lipoteichoic acid for attachment
(2) Hyaluronic acid capsule that inhibits phagocytosis
(3) Erythrogenic toxin causing the rash of scarlet fever
(4) Streptokinase, streptodornase (DNase B), and streptolysins.

Initially the lesion begins as a papule that rapidly evolves into a vesicle and to pustule that gradually enlarge and then break down over a period of 4 to 6 days to form characteristic thick crusts. The lesions heal slowly with depigmentation.

Streptococcal impetigo occurs on exposed areas of the body, most commonly seen on the lower extremities or face. Although regional lymphadenitis may occur, systemic symptoms are not usually present.

Anti-streptolysin O (ASO) antibodies are of no value in the diagnosis and management of impetigo because the activity of streptolysin O is inhibited by cholesterol, so the response of ASO is weak. In contrast, anti–DNase B levels are elevated in patients who are suspected of having post-streptococcal glomerulonephritis.[58]

Almost all staphylococci are penicillinase producers, so treatment with penicillin, now fails frequently.[59,60]

3. ***Pseudomonas***: They are Gram-negative bacilli which belongs to the family Pseudomonadaceae. These organisms are obligate aerobes and grow best in ambient air at 37°C, though they can grow at temperatures as high as 42°C.[61]

It has typical morphology of its colonies with metallic sheen, blue-green pigment, and a unique grape-like, fruity odor. The pigment pyoverdine, which is greenish-yellow and fluoresces under Wood's light, is common to the fluorescent group of Pseudomonas species (notably *P. aeruginosa, P. fluorescens, P. putida*), but pyocyanin, which is non-fluorescent and greenish-blue, is unique to *P. aeruginosa* and imparts the characteristic color.[62]

It has a positive oxidase test, and is unable to ferment carbohydrates but has the ability to grow on cetrimide agar. Growth at 42°C remains a defining characteristic of *P. aeruginosa* from the other fluorescent *pseudomonas*.[61]

P. aeruginosa has a plethora of features which contribute to its ability to cause disease. Sato et al [63] showed that strains of *P. aeruginosa* which were deficient in flagella and pili were unable to establish infection. Various proteases facilitate its dissemination by disrupting the basement membrane.[64] Other virulent factors include- phospholipase C,[65] ferri-pyochelin-binding protein,[66] lipopolysaccharide production,[67] and the products secreted by the type III secretion system.[68]

Green nail syndrome or chloronychia is the greenish discoloration of the nails that arises due to the pyocyanin pigment produced by the bacterium. It is characterized by the triad of green discoloration of the nail plate, proximal chronic paronychia, and disto-lateral onycholysis.

‘Hot tub folliculitis’ is caused by *P. aeruginosa* which is due to the recreational use of hot tubs, whirlpools, and swimming pools. It typically occurs in otherwise healthy individuals who are exposed to contaminated water. It presents as sudden onset of multiple, large, monomorphic, painful papules and pustules which occurs approximately 24 hours after the prolonged immersion in contaminated water. The lesions are clustered on body areas which are in contact with the water surface, especially the upper trunk, axillary folds, and buttocks. It is more common after immersion in a body of water with temperature over 38°C.

Ecthyma gangrenosum is a necrotic cutaneous lesion that is associated with *P. aeruginosa* bacteria. It is mostly found in immunocompromised individuals, although it can also develop in otherwise healthy individuals as a sign of underlying pseudomonal septicemia and can sometimes lead to fatal consequences.

4. ***E. coli***: It is a non-spore-forming, motile, Gram-negative bacterium, member of the family Enterobacteriaceae, which includes, facultatively anaerobic rod-shaped bacteria. They do not produce the enzyme oxidase*. E. coli* is 1.1–1.5-μm-wide, 2–6-μm-long and occur as single straight rod. It consists of flagella for motility and other appendages such as fimbriae or pili, which are proteinaceous structures that extend outward from the surface and help in attachment to surfaces including other cells or host tissues.

Treatment:

The choice of antibiotic for the treatment of skin and soft tissue infections is strongly influenced by the organisms that cause the infection and their antibiotic resistance profiles. It is also important to assess the wider impact of the chosen antibiotic on the development of antibiotic-resistant organisms, which could significantly compromise future treatment for that particular patient and the community as a whole. In children, most of the pyodermas are caused by either *S. aureus* or β-hemolytic streptococci.[47]

The antibacterial chosen must, therefore, target both these organisms to ensure high cure rates.

The discovery of penicillin in the 1940's led to a dramatic decrease in mortality due to staphylococcal infections, but penicillin-resistant strains that produced β-lactamase became common place in hospitals within a decade and, presently only <5% *S. aureus* strains are sensitive to penicillin.[47] Similarly, erythromycin resistance was reported to be >50%. Because of the extensive and inappropriate overuse of macrolides for the empiric treatment of infections, erythromycin resistance in *staphylococci* has paralleled the exponential rise in erythromycin resistance among β-hemolytic streptococci.

1. **Penicillin**: Penicillin was discovered in 1929, when Alexander Fleming noticed that a plate accidentally contaminated with penicillium mold lysed colonies of *staphylococci*. By 1940 significant quantities of the first penicillin from *Penicillium notatum* were cultured; however, it was not until in 1949 that penicillin was available for routine clinical use.[69]

There are five subclasses of Penicillin's, the natural, penicillin-resistant, amino, extended spectrum, and the anti-pseudomonal Penicillin's that combine β - lactamase inhibitors.

β -Lactam drugs damage the bacterial cell walls, thus interfering with synthesis. The three essential steps which are involved in interference with cell wall synthesis are: -

(1) attachment of penicillin to the drug receptors present on the bacteria, called penicillin-binding proteins (PBP),
(2) blockage of transpeptidation of peptidoglycan which inhibits cell wall synthesis, and
(3) activation of autolytic enzymes in the cell wall ultimately leading to bacterial demise.[70]

The drug of choice for *S. pyogenes* is Penicillin. It is safe and inexpensive drug, but has narrow spectrum. The penicillinase resistant Penicillin remains the antibiotic of choice for the treatment of penicillin-resistant, methicillin-sensitive staphylococcal infections because they are bactericidal with only few adverse effects. This group of antibiotics includes- cloxacillin, flucloxacillin, oxacillin, methicillin, nafcillin, and dicloxacillin. They all have similar pharmacokinetic profiles, with little difference in efficacy. The combination of different β-lactam labile Penicillin's with β-lactamase inhibitors such as -clavulanic acid, sulbactam, and tazobactam provides a new generation of antibiotics against MSSA. The group D enterococci are resistant to Penicillin's as well as penicillinase-resistant Penicillin's. Group B streptococci are resistant to tetracyclines but remain sensitive to the clinically achievable blood levels of penicillin. Sulbactam/ampicillin is another antibacterial combination which has been well tolerated in children.

The most common adverse effects of penicillin are nausea, vomiting, and diarrhea followed by drug fever, hemolytic anemia, neutropenia, interstitial nephritis, and a diffuse maculopapular erythematous eruption. Rare adverse effects include erythema multiforme, Stevens Johnson syndrome, toxic epidermal necrolysis, fixed drug eruption, vasculitis, erythroderma, pemphigus, acute generalized exanthematous pustulosis and anaphylaxis. The most common dermatologic adverse effect related to penicillin is a diffuse erythematous maculopapular eruption that starts on the trunk and spreads to the extremities and usually spares the palm, soles, and mucous membranes. These skin changes can occur one hour following drug administration or upto 10 days.

2. **Cephalosporins**: In 1945, Giuseppe Brotzu isolated the mold *Cephalosporium acremonium* from sewage that produced substances that inhibited both gram-positive and gram-negative bacteria.[71] They act by binding to penicillin-binding proteins and inhibit mucopeptide synthesis in

the bacterial cell wall. Cephalothin, the first cephalosporin, was marketed in 1964.

Cephalosporins are classified into generations based on their spectrum of activity; however, individual antibiotics may have marked differences in microbiological activity and other pharmacological properties.[71]

The first-generation agents are primarily active against gram-positive organisms including MSSA. They are also active against streptococci but not enterococci and have limited activity against gram-negative bacilli including *E. coli, P. mirabilis,* and *K. pneumoniae*. The drugs available include cefadroxil, cefazolin, and cephalexin.

Second-generation agents are active against gram-negative bacilli and have variable activity against gram-positive cocci. Cefamandole, cefaclor, and cefuroxime are the drugs available.

Third-generation cephalosporins are highly active against gram-negative bacilli, with some of these agents active against *P. aeruginosa* infections such as ecthyma gangrenosum. They are less active than first- and second-generation agents against gram positive cocci. They are the first line treatment for uncomplicated infections with *N. gonorrhoeae.*

Ceftriaxone and cefixime are first-line cephalosporins which are indicated for the treatment of adolescents, pregnant and lactating women as well as in the outpatient management of pelvic inflammatory disease.

Third-generation agents include ceftriaxone and cefotaxime, which are agents of choice for the treatment of *H. influenzae* cellulitis. *H. influenzae* causes a rare type of cellulitis of the face, neck, and upper extremities which occurs almost exclusively in young children under the age of 2 years.

Cefepime is the only fourth-generation cephalosporin, classified into a new class because of the extended spectrum of activity against both gram-

positive and gram-negative organisms including *P. aeruginosa* and is approved for use in skin and soft tissue infections.

Cephalosporins are active against *S. aureus, S. pyogenes*, *Proteus mirabilis, E. coli,* and *K. pneumoniae*. Higher-generation cephalosporins usually have broader spectrum of activity against Gram-negative organisms and are also effective against *H. influenzae* type b and some *Enterobacter* species. They are bactericidal, have excellent oral bioavailability and minimal hepatic metabolism.[72]

They are well tolerated by children because they have a pleasing taste and a few adverse effects only.[73] Adverse effects are nausea, vomiting, diarrhea, pruritus, rashes, urticaria, and erythema multiforme, although rare cases of arthralgia, thrombocytopenia, neutropenia, transient hepatitis, cholestatic jaundice, pseudomembranous colitis, anaphylaxis, and toxic epidermal necrolysis have also been reported.

Cefadroxil is at least as effective as cefalexin [74] and can be given once daily, but is more expensive among the second-generation cephalosporins. Cefprozil is as effective as the first-generation cephalosporins, with the added advantages of better tissue penetration, β-lactamase stability, fewer gastrointestinal adverse effects, and once daily administration.

Cefuroxime axetil is an oral prodrug that is activated in the intestinal mucosa and has increased absorption when taken with food.[75] It is effective in the treatment of uncomplicated skin and skin structure infections in children when given twice daily and has an activity similar to amoxicillin/clavulanic acid.[75] Parenteral cefuroxime is also useful in treating severe infections in children.

Cefixime has poor activity against *S. aureus* because of low-affinity for β-lactam binding proteins,[47,76] and has more gastro intestinal adverse effects, particularly diarrhea.[77] On the other hand, Cefpodoxime proxetil, a

prodrug like Cefuroxime axetil which is activated in the intestine and has excellent activity against *S. aureus*, and long serum half-life with excellent tissue penetration allows twice daily administration, and is well tolerated by pediatric patients.[78]

Cefdinir is third-generation cephalosporin with excellent activity against many Gram-negative and Gram-positive aerobic organisms, including *S. pneumoniae, S. aureus, S. pyogenes, H. influenzae,* and *Moraxella catarrhalis*.

Ceftriaxone is an intramuscular and intravenous third generation cephalosporin. It has long serum half-life allowing for once daily administration. It is active against *S. aureus* and *S. pyogenes*. It is effective in the treatment of skin and soft tissue infections in children, [79,80] making it the drug of choice for outpatient treatment of moderately severe skin infections, particularly in patients who are unable to tolerate oral antibiotics.

Ceftaroline is a novel “fifth-generation” cephalosporin approved for treatment of complicated MRSA by the FDA in 2010.[81] It has a rapid bactericidal effect, with a high affinity for the PBP2a, a MRSA specific PBP. It is a good empirical antibiotic for SSTIS, as it also has activity against coagulase-negative staphylococci, *enterococci*, many *streptococci*, and anaerobic organisms.[82] The recommended dose for SSTIs is 600 mg every 12 hours administered intravenously (IV) or intramuscularly (IM). Dosage adjustment is necessary for patients with renal impairment.[83] The most common adverse effects are nausea, diarrhea, headache, and pruritus.[84] Caution is necessary in patients with allergy to Penicillin’s or other beta-lactams, since cross-sensitivity may occur. It is classified as pregnancy category B drug. It has not been evaluated in pediatric populations.[83]

3. **Macrolides**: Erythromycin was the first macrolide antibiotic derived from *Streptomyces erytherus,* in 1952.[70] It is used in patients who are

allergic to Penicillin. All the preparations of erythromycin have the same spectrum of activity and uses.

Clarithromycin and Azithromycin have longer half-life, better tissue penetration, and an extended spectrum of activity, with increased gastric acid stability and fewer gastrointestinal adverse effects.[85]

Macrolides bind reversibly to the 50S ribosomal subunit of susceptible bacteria and inhibits RNA-dependent protein synthesis by preventing transpeptidation and translocation reactions.

Although generally bacteriostatic, clarithromycin and azithromycin are bactericidal against *S. pyogenes, S. pneumoniae, and H. influenzae*.[86] Macrolides are active against most streptococci and *S. aureus* but not against MRSA. Erythromycin-resistant *S. aureus* is not susceptible to the newer macrolides.

Erythromycin is effective in treating P. acnes, rosacea, and perioral dermatitis. Other effective uses for macrolides are erythrasma caused by *C. minutissimum*; erysipeloid caused by *E. rhusiopathiae*; anthrax caused by *B. anthracis*; and *B. burgdorferi*, the cause of Lyme disease.

Studies have shown that clarithromycin and azithromycin are equivalent to erythromycin in treating skin and soft tissue infections.[87,88] Another study found that cephalexin was equivalent to azithromycin.[89]

Azithromycin and clarithromycin are effective in treating cutaneous mycobacterial infections such as M. leprae.

The adult dose for erythromycin is 250-500 mg four times a day. Since the liver metabolizes the macrolides, renal adjustments are not necessary and extra doses following hemodialysis are not required. The usual adult dose of azithromycin is 500 mg for the first day of therapy followed by 250 mg once daily for a total of 5 days. The adult dose of clarithromycin is 250-500 mg twice a day.

The most common adverse effects of macrolides are nausea, vomiting, abdominal cramps, and diarrhea. Rarely, liver toxicity can manifest as cholestatic hepatitis, which presents as nausea, vomiting, and abdominal pain followed by jaundice and fever. Eosinophilia and raised liver function tests occur almost exclusively with the erythromycin estolate preparation.

Other adverse effects include photosensitivity and angioedema which occur with azithromycin. Cutaneous reactions with erythromycin are extremely rare.

4. **Fluoroquinolones**: The prototype antibiotic-nalidixic acid, was discovered accidently in 1960 during the synthesis of the antimalarial agent chloroquine.[90] It includes- trovafloxacin, levofloxacin, sparfloxacin, grepafloxacin, moxifloxacin, and gatifloxacin.

They are bactericidal and inhibit bacterial topoisomerase enzyme which is responsible for maintaining DNA in a biologically active state. There are two types of enzyme topoisomerase, type I and II. Fluoroquinolones inhibit type II topoisomerase, also known as DNA gyrase, the enzyme responsible for introducing negative supercoils, and also eliminate positive supercoils which occurs ahead of the DNA replication fork during DNA replication.[91]

Fluoroquinolones are active against a variety of gram-positive and gram-negative organisms, such as *S. aureus*, including MRSA, resistance has emerged because of its widespread use. Ciprofloxacin is a first-line agent for *B. henselae*, the causative organism of cat scratch disease. It is also active against *N. gonorrhoeae* and *C. trachomatis*. Ofloxacin is drug of choice for pelvic inflammatory disease.[92] Ciprofloxacin is recommended as a treatment option for the organism *Haemophilus ducreyi,* the etiologic agent responsible for chancroid. Nail infections caused by *P. aeruginosa* are sensitive to ciprofloxacin.

On 15 September, 2000 the FDA approved levofloxacin at a dose of 500 mg once daily for the treatment of complicated pyodermas. Recently FDA has approved levofloxacin at a dose of 750 mg once daily to be used for complicated skin infections.

The most common adverse effects include nausea, diarrhea, insomnia, headache, and dizziness. Dermatological side effects include morbilliform eruption, pruritus, vasculitis, leg pigmentation, urticaria and phototoxic reactions that can occur even with concomitant use of sunscreens. Thus, patients should be recommended to avoid direct sun exposure while using fluoroquinolones.

Hepatic side effects include transient increases in transaminases, cholestatic jaundice, hepatitis, and hepatic failure. Blood abnormalities include leukopenia. Musculoskeletal adverse effects include arthropathy, tendonitis, and tendon rupture. Cardiovascular complications include hypotension, tachycardia, and prolongation of QTc interval. Renal adverse effects are azotemia, crystalluria, hematuria, interstitial nephritis, renal failure, and nephropathy. Headache, dizziness, insomnia, mood changes, confusion psychosis, tremors, and seizures are central nervous system adverse effects. Other side effects are drug fever, chills, serum sickness-like reaction, angioedema, bronchospasm, and anaphylaxis. Pregnant and nursing mothers and children should not receive fluoroquinolone therapy.

5. **Oxazolidinones**: **Linezolid:** - On 18 April, 2000, the FDA approved linezolid, the first antibiotic in this new class that has dermatologic application. It inhibits early protein synthesis by inhibiting the initiation complex at the 30S ribosome.[93,94]

The spectrum of activity is similar to vancomycin.[95] It is active against gram-negative anaerobes and mycobacteria.[96] It has bacteriostatic activity against enterococci and staphylococci and bactericidal activity against *S. pyogenes* and *Bacteroides fragilis*.[97] It is approved for the

treatment of gram-positive pneumonia, vancomycin-resistant *Enterococcus faecium*, and complicated skin infections caused by MRSA.

It is available in 600-mg tablet, an oral suspension containing 100 mg/mL, and an IV solution. It is 100% bioavailable orally so patients may be switched from intravenous to oral preparation without a dose change. Tedizolid can be administered once daily, either orally or IV.

The most common adverse effects are diarrhea, headache, nausea, vomiting, leukopenia, anemia, pancytopenia, thrombocytopenia, and elevated liver and renal function tests. Following discontinuation, hematologic parameters return to pre-treatment levels. It is a pregnancy category C drug.

6. **Clindamycin**: It is a chlorine-substituted derivative of lincomycin, an antibiotic derived from *Streptomyces lincolnensis*.[70] It is a bacteriostatic antibiotic that inhibits protein synthesis by blocking 50S ribosomal subunit.

It is most frequently used in dermatology as an alternative for treating streptococcal and staphylococcal infections in the penicillin-allergic patients and for treating most of the anaerobic bacteria including *P. acne*. Other indications include treatment of paronychia caused by *S. aureus*. High levels of the drug are achieved in the nasal secretions; therefore, it is the most effective agent for eradicating nasal carriage of *S. aureus* and prevents recurrent staphylococcal infection.

The recommended adult dosage is 150-300 mg po four times a day; intravenous and oral preparations are available. For preventing recurrent staphylococcal infections, clindamycin 150 mg once a day for 3 months is an acceptable regimen.[98]

It is metabolized by liver; thus, renal adjustments and extra doses following hemodialysis are not necessary. Caution must be exercised in treating patients with hepatic failure.

The most common side effect experienced by patients is diarrhea. In hospitalized patients, C. difficile colitis may develop in patients treated with clindamycin or broad-spectrum antibiotics since the normal intestinal bacteria flora is often eradicated. Fever, leukocytosis, and occasionally intravascular volume depletion can accompany the diarrhea. Therefore, patients with inflammatory bowel disease should not receive clindamycin.

The common dermatological side effect is morbilliform rash. Less common side effects include anaphylaxis, urticaria, and erythema multiforme.

7. **Trimethoprim-Sulfamethoxazole** (TMP-Sulfa): In 1932, a red dye-prontosil, was synthesized by two German chemists.[70] The compound was ineffective in vitro, but several years later another German noted that, in vivo, the compound was converted to sulfanilamide. It was active against hemolytic streptococcal bacteria. The synergistic addition of trimethoprim to sulfamethoxazole has made the folate inhibitor more popular and effective. Sulfonamides are structural analogs of p-aminobenzoic acid (PABA).

Sulfonamides are competitive inhibitors of enzyme dihydropteroate synthetase. Trimethoprim inhibits the enzyme dihydrofolate reductase, the next sequential step in nucleic acid metabolism. Sulfonamide action is bacteriostatic and reversible by removal of the drug or by the presence of excess PABA.

Trimethoprim-sulfamethoxazole is active against a variety of gram-negative and gram-positive organisms, including Pneumocystis carinii, Shigella, Salmonella, and Chlamydia trachomatis, and against *S. aureus* including MRSA. Trimethoprim-sulfamethoxazole is used mainly to treat uncomplicated urinary and respiratory tract infections. Trimethoprim-sulfamethoxazole has limited dermatological indications.

It has been indicated for the treatment of acne refractory to tetracycline and Wegener's granulomatosis.[70]

Intravenous and oral preparations are available. The normal oral adult dose is 800 mg of sulfamethoxazole and 160 mg of trimethoprim taken twice daily.

It can cause megaloblastic anemia, leukopenia, granulocytopenia, and hemolytic anemia in patients with glucose-6-phosphate dehydrogenase deficiency.[70] It is also not recommended for patients with renal insufficiency since nephrotoxicity can occur. Other common adverse effects include drug reactions and gastrointestinal side effects such as nausea, vomiting, and abdominal discomfort.

In HIV-infected patients, thrombocytopenia and neutropenia are common adverse effects. Furthermore, 30%-85% of HIV patients develop morbilliform eruptions, mostly during the second week of therapy, compared with a similar rash that develops in 3 % of the general population.[99]

8. **Rifampicin**: Rifampicin and rifabutin are rifamycins. Rifampicin is produced by *Streptomyces mediterranei*. Rifampin is well absorbed orally and penetrates phagocytic cells and eradicates intracellular as well as extracellular bacteria and mycobacteria.

Metabolism of rifampicin occurs in the liver and absorption is impaired if taken following a meal. Excretion is via bile and ultimately the feces. Rifampicin inhibits DNA-dependent RNA polymerase in susceptible bacteria and chlamydia.

It is bactericidal against *staphylococci, N. meningitidis, N. gonorrhoeae,* and *H. influenza*. It has poor gram-negative coverage. When combined with TMP-Sulfa, it eradicates staphylococcal nasal carriage.

Its indication in dermatology is to treat cutaneous tuberculosis and is the only drug bactericidal to M. leprae. Cutaneous leishmaniasis and rhinoscleroma are also effectively treated with rifampin.

Adverse effects include- hepatotoxicity, thrombocytopenia and nephritis. It imparts a harmless orange discoloration to urine, sweat, and tears, including contact lenses. High-dose intermittent therapy leads to a hypersensitivity syndrome which is characterized by fever, chills, headache, dizziness, bone pain, pruritus, urticaria, acneiform eruptions, bullous pemphigoid-like lesions, mucositis, exfoliative dermatitis, and exudative conjunctivitis associated with eosinophilia.[100]

It is a potent enzyme inducer of the cytochrome P450. It can lead to reduced efficacy of contraceptives and anti-inflammatory properties of glucocorticoids.

9. **Metronidazole**: Oral metronidazole is the drug of choice in the treatment of bacterial vaginosis (BV), trichomonas vaginalis, intestinal Giardia, and amoebiasis infections. It has a broad spectrum of activity against protozoal organisms, anaerobes including Bacteroides spp., and clostridia.

Since metronidazole is metabolized in the liver, dose adjustment may be required for patients with hepatic insufficiency.

Its mechanism of action in the skin is unknown but it is suspected to impede leukocyte chemotaxis and also have antibacterial and antioxidant as well as anti-inflammatory properties.[101,102]

Adverse effects include nausea, diarrhea, stomatitis, and peripheral neuropathy with prolonged use. Alcohol must be avoided because of disulfiram-like effect.

10. **Aminoglycosides**: Aminoglycosides were isolated from the Streptomyces species. They include- amikacin, gentamicin, netilmicin, spectinomycin, streptomycin, tobramycin, and neomycin. They inhibit protein synthesis by binding to the 30S ribosomal subunit. All are potentially ototoxic and nephrotoxic, thus, dose adjustments in renal failure is required.

They are most commonly used to treat gram-negative enteric bacteria. Dermatologic indications include-Pseudomonas septicemia with gentamicin, tobramycin, and amikacin. It is combined with an anti-Pseudomonas penicillin or ceftazidime in acutely ill patients.

11. **Dapsone**: In 1940, sulfapyridine was first described following successful treatment of dermatitis herpetiformis. In the early 1950s, dapsone, a chemical analog of sulfapyridine, was introduced.

Dapsone is well tolerated and absorbed from the gastrointestinal tract and is retained in skin, muscle, liver, and kidney. The most common adverse effects include fever, fatigue, headache, nausea, anorexia, pruritus, and gastrointestinal intolerance followed by hypoalbuminemia, psychosis, leukopenia, agranulocytosis, hemolysis, methemoglobinemia, peripheral neuropathy, hepatitis, infectious mononucleosis, and cholestatic jaundice. Dermatological adverse effects include morbilliform eruptions, erythema nodosum, erythema multiforme, and exfoliative dermatitis.

12. **Tetracyclines**: It was discovered in 1948 when chlortetracycline was isolated from *Streptomyces aureofaciens*.[70] Subsequently, catalytic dehalogenation of chlortetracycline yielded tetracycline in 1953.

Although there are three subclasses of tetracycline, only two are commonly used by dermatologists: the short-acting tetracycline and the long-acting doxycycline monohydrate and minocycline. The intermediate-acting methacycline and demeclocycline are not commonly used. Tetracyclines are the prototype broad-spectrum antimicrobial agent. They are bacteriostatic for many gram-positive and gram-negative bacteria including some anaerobes, rickettsiae, chlamydiae, and mycoplasma, as well as some protozoa and amoebas.

Tetracyclines are bacteriostatic antibiotics that interferes with the 30S ribosomal subunit, inhibiting protein synthesis.

The normal adult dose of tetracycline is 250-500 mg four times a day. The most common adverse effects include gastrointestinal complaints like nausea, vomiting, and diarrhea. C. difficile colitis is a complication of tetracycline therapy more common in hospitalized patients. It can lead to azotemia; therefore, not recommended in patients with renal failure.

13. **Glycopeptides**: Vancomycin is the first-line antimicrobial therapy for MRSA.[24] The increasing reports of MRSA treatment failure with vancomycin, and the development of vancomycin-resistant *S. aureus* (VRSA) are an indication that the efficacy and utility of vancomycin is decreasing. Recent studies suggested that greater microbiological cure rates in a shorter period of time can be achieved with alternatives to vancomycin, such as teicoplanin.[103]

It can be administered intravenously to treat infections caused by gram-positive bacteria, including MRSA and methicillin-resistant coagulase-negative staphylococci. Teicoplanin is bactericidal, has a longer half-life and is less nephrotoxic as compared to vancomycin.[104,105] The oral formulation of teicoplanin is not absorbed, but is an effective treatment for *C. difficile* gastrointestinal infections. Like vancomycin, teicoplanin is pregnancy category C.

The risk factors for Vancomycin-associated nephrotoxicity are- longer hospital stays, increased medical costs, need for additional antibiotics, and, in rare cases, dialysis.[106,107] Vancomycin-resistant *Enterococcus* (VRE) species have been reported.[108,109]

Telavancin is another member of the lipoglycopeptide class and is a semisynthetic derivative of vancomycin.[110] It exhibits activity against a broad range of gram-positive organisms, including *S. aureus*, coagulase-negative staphylococci, and *Streptococcus* species. It has activity against MRSA, vancomycin-intermediate *S. aureus*, vancomycin-susceptible and VRE species, and various other gram-positive anaerobic organisms.[110]

14. **Cyclic Lipopeptides**: Daptomycin is a parenteral lipoprotein with potent bactericidal activity against gram-positive bacteria, including MRSA and VRE.[111] It has clinical efficacy comparable with vancomycin.[112]

Daptomycin's mechanism is somewhat unique and distinct. It binds to bacterial membranes, forming channels within the cell wall, allowing efflux of potassium, and causing rapid depolarization of the bacterial membrane.[113] Hence, cross-sensitivity with other drugs is practically non-existent.[114]

The most notable side effect of daptomycin is myotoxicity, which is associated with elevated serum creatine phosphokinase and creatine kinase and is reversible upon discontinuation of therapy.[115-117] It should be discontinued if muscle pain or weakness develop, or if creatine kinase levels rise 5- to 10-fold. It is a pregnancy category C drug.[116]

15. **Lipoglycopeptides**: Telavancin, dalbavancin, and oritavancin are semi-synthetic lipoglycopeptides that inhibit bacterial cell wall synthesis.[118] All three agents target gram-positive organisms, including MRSA and vancomycin-intermediate *S. aureus* (VISA), but only oritavancin is active against VRSA.[119]

Compared with vancomycin, all three have longer half- lives, allowing for once-daily dosing for telavancin, once-weekly dosing for dalbavancin, and perhaps just one dose per treatment course for oritavancin.[120]

The most common adverse effects reported with telavancin are nausea, taste disturbance, and insomnia, and with dalbavancin are fever, nausea, diarrhea, and constipation, and with oritavancin are injection site reactions, fever, and diarrhea.[121,122]

Telavancin was approved by FDA in 2009 for the treatment of complicated MRSA skin and skin structure infections.[123-125]. Telavancin is a pregnancy category C drug.[126,127] Dose adjustments are required in patients with renal failure.[128,129]

16. **Glycylcyclines**: Tigecycline is a novel derivative of minocycline, which binds to the 30s ribosomal sub-unit. The drug is effective against gram-positive, gram-negative, anaerobic, and atypical organisms and MRSA, VRE.[130] Tigecycline dosing is 100 mg IV loading dose, followed by 50 mg IV every 12 hourly. The drug is well tolerated with infrequent adverse effects such as -nausea, vomiting, and headache. Intravenous tigecycline has been recently linked to increased mortality compared with other drugs with the same indication.[131,132]

17. **Streptogramins**: The streptogramin combination of Quinupristin - Dalfopristin has bactericidal activity against a wide array of complicated gram-positive organisms, including most MRSA, MSSA, and streptococci strains.[133,134] The drug is administered via central venous catheter to offset thrombophlebitis at the infusion site. Other side effects include arthralgias, myalgias, hyperbilirubinemia, hepatic toxicity, and QTc prolongation.[135] It is a pregnancy category B drug.[133]

INVESTIGATIONAL AND RECENTLY APPROVED ANTIBIOTICS FOR ABSSSI:

1. **Aminomethylcyclines**: Omadacycline

Omadacycline is a first-in-class broad-spectrum aminomethylcycline.[136] Similar to tetracyclines, omadacycline binds to the 30S ribosomal subunit of target gram-positive and gram-negative bacteria, resulting in inhibition of protein synthesis.[136]

Omadacycline has high potency against MRSA and MSSA and maintains activity in the presence of ribosomal protection and efflux tetracycline resistance genes. It has proven activity against tetracycline-resistant pathogens and is not affected by mechanisms of resistance to other classes of antibacterial agents.[136,137]

2. **Pleuromutilins**: Lefamulin

Lefamulin is currently in late stage development as an IV and oral treatment for ABSSSIs.[138] Lefamulin inhibits protein synthesis by binding to the 50S ribosomal subunit.[139] It exhibits activity against gram-positive organisms, including MRSA, MSSA, *S. pyogenes*, *S. agalactiae*, and vancomycin-resistant *Enterococcus faecium*.[140]

They are not affected by resistance mechanisms involving major antibiotic classes, including fluoroquinolones and tetracyclines.[138]

Topical Antibiotics:

Topical antibiotics are most frequently used by dermatologists to treat localized infections like impetigo and are also used as prophylactic agents to prevent infection following abrasions. Topical agents are not absorbed systemically; therefore, the likelihood of toxicity is very low.

Topical antiseptics are commonly used on the intact skin to diminish the risk of bacterial contamination or infection. These agents are used prior to a surgical procedure or for cleaning minor cuts and abrasions. They are effective for two reasons: first, they have direct antibiotic effects and, second, the anti-inflammatory properties suppress neutrophilic chemotactic factors.

1. **Topical mupirocin:** It is derived from *Pseudomonas fluorescents*. Antimicrobial activity is limited to gram-positive organisms including *staphylococci* and *streptococci*. Gram-negative bacilli are resistant to it.

The ointment and cream are available in a 2% preparation and should be applied three times a day for the localized treatment of bacterial infections such as non-immunocompromised cases of impetigo. For nasal MRSA eradication, mupirocin nasal ointment 1 g divided between nostrils twice daily for 5 days is available in single-use 1-g tubes.

2. **Fusidic acid**: Its 2% cream is used for treatment of localized pyoderma up to 3 times daily for up to 10 days. As compared to mupirocin, it is 40% to 80% more cost effective.

3. **Retapamulin:** It is a semi-synthetic agent and is derived from an edible mushroom called *Clitopilusscyphoides*. It inhibits protein synthesis by binding to bacterial ribosomes. It is effective against *S. aureus* and *S. pyogenes*, but is not indicated for MRSA infections. It is available as a 1% ointment and can be used in children older than 9 months of age.

4. **Topical gentamicin**: It is an aminoglycoside, is available as topical 0.1% cream or ointment. It is used as a prophylaxis against malignant otitis externa caused by gram-negative *P. aeruginosa* in diabetics and immunocompromised patients.[141,142]

5. **Topical neomycin sulfate**: It is an aminoglycoside ointment used to treat infections caused by gram-negative bacilli. Neomycin binds to the 30S ribosomal subunit to inhibit protein synthesis.

It is used mostly as a prophylaxis against infections in superficial abrasions, cuts, and burns and is commonly mixed with other antibiotics to create combination agents.[143,144]

6. **Topical bacitracin**: It is a cyclic polypeptide with components A, B, and C. Bacitracin A is available as a zinc salt and is the most commonly available formulation. Bacitracin interferes with bacterial cell wall synthesis and is active against gram-positive cocci. Resistance is common among most gram-negative and yeast organisms. It is indicated for the treatment of superficial bacterial infections of the skin such as impetigo, furunculosis.[145]

Polymyxin B and neomycin are combined with bacitracin to form a triple antibiotic preparation indicated for the treatment of secondarily infected atopic, nummular, and stasis dermatitis.

Contact allergy to bacitracin is a relatively uncommon occurrence. However, patients with stasis dermatitis are commonly sensitive to bacitracin.

7. **Topical polymyxin B**: It is a cyclic polypeptide cationic detergent in ointment form that disrupts cell membrane integrity. Contact allergy to polymyxin B is rare. It is active against gram-negative organisms such as *P. aeruginosa, Enterobacter*, and *E. coli*.

A solution containing polymyxin or neomycin is sometimes used in combination with a steroid to decrease inflammation and infection associated with otitis externa, also known as 'swimmer's ear'.[146]

8. **Topical gramicidin:** It is an antibiotic derived from *B. brevis* that forms stationary ion channels in susceptible gram-positive bacteria.

9. **Topical chloramphenicol:** It is an antibiotic derived from *S. venezuelae* used to treat minor bacterial infections. The topical 1% cream is rarely used because of reported fatal aplastic anemia or dose-related bone marrow suppression following topical exposure.

10. **Topical silver sulfadiazine**: It is a broad-spectrum sulfonamide antibiotic cream used commonly in burn patients. Its acts by slow release of silver into the tissue.

11. **Topical nitrofurazone**: It is available as a 0.2% cream, solution, and soluble dressing used to treat burn patients. It is active against *staphylococci, streptococci, E. coli, C. perfringens, Aerobacter enterogenes, and Proteus* species.

12. **Topical Nadifloxacin:** It is a newer topical fluoroquinolone antibacterial compound with a benzoquinoline skeleton with fluorine at the sixth position and N-hydroxypiperidine at the eighth position. It has broad-spectrum of activity against Gram-positive bacteria, including coagulase-negative *Staphylococcus* species and *Propionibacterium*

acnes granulosum. It is also very effective against Gram-negative bacteria including *P. aeruginosa* and *E. coli*.[147]

S. aureus colonizes anterior nares in 20% to 40% of normal adults. Eliminating it from the nares may help reduce the frequency of attacks in patients suffering from recurrent staphylococcal pyodermas and may help terminate the spread of organisms in nosocomial staphylococcal epidemics. Topical antibiotics have varying success in this situation. Topical gentamicin eliminates the carriage state in 65%[141] to 100%,[142] bacitracin-neomycin in 72%,[143] neomycin-chlorhexidine in 63%[144] to 75%,[141] bacitracin in 38%,[145] vancomycin in 25%[145] to 42% , oxacillin in 88%,[146] and a combination of neomycin, gramicidin, polymyxin B, and hydrocortisone in 89%.[146]

Mupirocin is the most effective topical antibiotic.

INFECTIONS IN THE IMMUNE COMPROMISED HOST

In immunocompromised patients there is a decreased ability to control local infection, so these patients are at increased risk of skin infection.[148] Infection prevention in such patients is important and requires careful attention to protect the skin from unnecessary trauma, maceration, or alterations in the normal microbial flora. When infections do develop, it is critical to establish a specific etiological diagnosis, because many are nosocomial and are caused by resistant pathogens.

Patients with neutropenia are predisposed to infection because of insufficient circulating neutrophils, lack of adequate myeloid marrow reserve and congenital or acquired defects in function of neutrophils.[148,149]

Cutaneous manifestations include erythematous maculopapular rash, focal or progressive cellulitis, cutaneous nodules and ecthyma gangrenosum. The most commonly isolated organisms are *Pseudomonas* species, *Aeromonas* species, *Serratia* species*, S. aureus, Stenotrophomonas maltophilia*.

Gram-positive organisms are most frequently isolated pathogens because of the increased use of antimicrobial prophylaxis with fluoroquinolones or trimethoprim-sulfamethoxazole and the frequent reliance on indwelling vascular access devices. These organisms, in order of decreasing prevalence include coagulase-negative staphylococci, *streptococci viridans, enterococci, S. aureus, Corynebacterium* species, *Clostridium* species, and *Bacillus* species and often represent part of the patient's normal skin flora. Soft tissue infections due to these pathogens usually begin as a focal area of erythematous cutaneous tenderness, a macular or maculopapular rash, or cellulitis.

Prevention

Preventive educational information on personal hygiene and appropriate wound care are recommended for patients with skin or soft tissue infections (SSTI) especially those with recurrent furunculosis.[150,151] Wounds should be covered with clean, dry bandages and good personal hygiene with regular bathing and washing of hands with soap and water, or cleansing with an alcohol-based hand gel is suggested.

Avoid sharing personal items like disposable or electrical razors and epilators, linens, and towels.

Frequently touched surfaces such as- counters, door knobs, bath tubs, and toilet seats, should be regularly cleaned. Commercially available cleaners or detergents appropriate for the surface being cleaned should be used in keeping with product instructions for routine cleaning of surfaces.

Colonization

Decolonization with topical mupirocin and chlorhexidine can reduce the incidence of subsequent *S. aureus* infections. It can be considered in cases with recurrent pyodermas and in cases with ongoing transmission among household members or other close contacts despite hygiene measures. It consists of application of mupirocin ointment twice daily within the nostrils for 5-10 days and daily body wash with 4% chlorhexidine soap for 5 -14 days. Dilute bleach baths 15 minutes, twice daily for 3 months can be done. Oral rinsing with 0.2% chlorhexidine solution three times daily decreases pharyngeal flora.[152] Topical gentian violet 0.3% solution applied to the nostrils twice daily for 2 to 3 weeks has also been suggested.[153]

Oral antimicrobial therapy is not routinely recommended for decolonization. It is recommended for the treatment of active infection only.

If family members are carriers they should be treated as the patient. Nutritional status of patient should be improved.

Rectal swab cultures may be considered in refractory cases since the gastrointestinal tract may be a reservoir of MSSA and MRSA. In these cases, oral vancomycin (1 g twice daily for 5 days) can eradicate 80%–100% of MRSA gut colonization.

Vaccine

Vaccine development for *S. aureus* has been stymied by multiple factors, including lack of understanding of human immunity to staphylococci, redundancy of virulence determinants within the staphylococcal genome, and failure of previous vaccine candidates.

In a study, the vaccine adequately elicited anti-capsular antibodies but failed to protect recipients from clinical disease. This vaccine has been

modified significantly to include other targets and is currently in clinical development.[154]

Despite such failures, staphylococcal vaccine development, as well as strategies for passive immunization still continues. Once available, a suitable vaccine will be targeted to individuals at high risk for SSTI and those with recurrent infections.[155]

MATERIALS AND METHODS

Source of Data

Patients who attended outpatient and inpatient Department of Dermatology of Dayanand Medical College and Hospital, Ludhiana, were taken as the study material and the source of data.

Method of Collection of Data

All clinically diagnosed cases of pyoderma attending outpatient and inpatient Department of Dermatology of Dayanand Medical College and Hospital, Ludhiana, were included in this study. Detailed history of the patients including duration of disease, site of infection, symptoms related to the disease was taken. Pus sample was collected and processed in the Department of Microbiology. Gram staining, and Culture and sensitivity of all the samples was done. For sensitivity, OPD samples were processed manually while IPD samples were processed by automated method as per institutional protocol.

The following data was collected-

- Patient's details including name, age, sex, MRD no., history (past, personal, family, treatment)
- Date of collection of samples
- Local examination findings
- Provisional clinical diagnosis
- Gram's stain findings
- Pus culture and sensitivity report

Inclusion Criteria:

A total of two hundred clinically suspected cases of pyoderma attending outpatient and inpatient Department of Dermatology of Dayanand Medical College and Hospital, Ludhiana, were included in this study.

Exclusion Criteria:

1. Patients not willing to participate
2. Patients already on Antibiotic therapy
3. Patients who took any antibiotic in last 7 days

Number of cases

Two hundred

Procedure of sample collection and transportation

Pus samples were collected with the sterile cotton tipped swabs/syringes.

Specimen transportation

Samples were collected and immediately transported to Microbiology laboratory

Processing of specimens

All the samples received in the Microbiology department were processed as per standard protocol.[156]

Direct Microscopy:

Smears were prepared from pus samples. Gram staining was done.

Gram's staining [156]:

1. A clean glass slide was taken and smear was prepared.
2. The smear was air dried and fixed by flaming.
3. The slide was placed with smear upwards over a staining rack.
4. Smear was covered with gentian violet which was left to act for one minute.
5. The slide was held at a steep slope with the help of forceps. Gram's iodine solution was poured from one end and the gentian violet stain was washed off.
6. The smear was covered with fresh iodine solution and the slide was left horizontally on the rack for one minute.

7. Decolorization of the smear was done with acetone and immediately washed with water.
8. Slide was covered with 0.5% safranine for 30 seconds.
9. It was washed thoroughly with water, dried and seen under oil immersion lens of the microscope.

Culture

Pus samples were inoculated by a sterile inoculating loop on Blood agar plate and Mac Conkey's agar plate within an hour of receiving the sample. Culture plates were incubated at 35-37°C for 24-48hrs.

Identification-

Isolates were identified on the basis of Gram staining, colony characteristics, motility, various biochemical reactions such as catalase, coagulase, indole, methyl red, oxidase, citrate, urease, phenyl pyruvic acid, voges-proskauer test.[156]

Antimicrobial susceptibility testing:-

Antimicrobial susceptibility testing was done using Kirby Bauer Disc diffusion method using Muller Hinton agar as per CLSI guidelines.[157]

Automated identification and antimicrobial susceptibility testing:-

Growth was identified by colony characters, gram staining and Vitek-2 system.

For gram positive organisms, the antimicrobial agents tested were:

Penicillin (10µg), amoxicillin / clavulanic acid (20/10µg), clindamycin(2µg), amikacin (30µg), gentamycin (10µg), cotrimoxazole (1.25/23.75µg), ciprofloxacin (5µg), erythromycin (15µg), cephalexin (30µg), linezolid (30µg), vancomycin (30µg), teicoplanin (30µg).

For Gram negative organisms- amikacin (30µg), ampicillin(10µg), gentamycin (10µg), ciprofloxacin (5µg), cotrimoxazole (1.25/23.75µg), cefotaxime (30µg), piperacillin (100µg), piperacillin + tazobactam

(100/10µg), cefoperazone+ sulbactam (75/30µg), imipenem (10µg), polymyxin B (300µg).

Antimicrobial discs were used for quality control- *E. coli* ATCC (25922), *Pseudomonas* ATCC (27853) and *S. aureus* (25923) were used as control organisms as per CLSI guidelines.[157]

Interpretation:-

The diameter of zone of inhibition was measured and interpreted as susceptible, intermediate or resistant.

For detection of MRSA cefoxitin disc diffusion technique was used as per CLSI guidelines.[157]

Statistical analysis:

Data was described in terms of range; mean ± standard deviation (± SD), median, frequencies (number of cases) and relative frequencies (percentages) as appropriate. For comparing categorical data, Chi square ($\chi 2$) test was performed and exact test was used when the expected frequency is less than 5. A probability value (p value) less than 0.05 was considered statistically significant. All statistical calculations were done using SPSS (Statistical Package for the Social Science) SPSS 21 version statistical program for Microsoft Windows.

OBSERVATIONS AND RESULTS

The present study included 200 clinically diagnosed cases of pyoderma, attending the outpatient and inpatient Department of Dermatology, Dayanand Medical College and Hospital, Ludhiana. The study was conducted from 1st June, 2019 to 30th May, 2020. Cases were divided on the basis of age into two groups. The first group consisted of 100 children of <18 years of age and second group consisted of 100 adults of >18 years of age.

After the informed consent, detailed history of the patient was taken, and clinical examination was done. For laboratory diagnosis, specimens of pus were collected and sent for gram staining and bacterial culture and sensitivity in the Microbiology department of DMCH. The observations and data obtained from the study were compiled and analyzed. All the results are depicted in the tables and graphs that follow.

DEMOGRAPHICAL DATA

Table 1: Distribution of patients.

OPD/ IPD	Child		Adult		Total		p-value
	No. of cases	Percentage	No. of cases	Percentage	No. of cases	Percentage	
OPD	96	53.30%	84	46.70%	180	90%	**0.008**
IPD	4	20.00%	16	80.00%	20	10%	
Total	100	50.00%	100	50.00%	200	100%	

180 (90%) patients were recruited from OPD and 20 (10%) were admitted in IPD.

Out of 180 (90%) patients who visited OPD, 96 (53.3%) were children and 84 (46.7%) were adults.

Out of 20 (10%) patients admitted in IPD, 4 (20%) were children and 16 (80%) were adults.

The distribution of patients visiting OPD and IPD in the two groups is statistically significant (0.008).

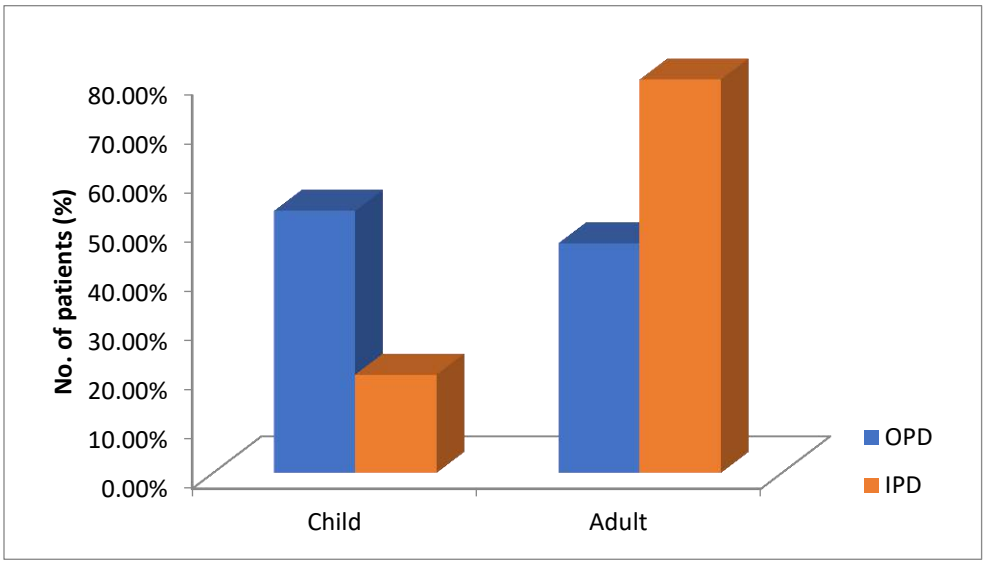


Figure 1: Distribution of patients.

Table 2: Age distribution of patients.

Age group	Child		Adult		Total	
(in years)	No. of cases	Percentage	No. of cases	Percentage	No. of cases	Percentage
<10	68	68%	0	0%	68	34%
10-18	32	32%	0	0%	32	16%
19-30	0	0%	21	21%	21	11%
31-40	0	0%	18	18%	18	9%
41-50	0	0%	21	21%	21	11%
51-60	0	0%	21	21%	21	11%
61-70	0	0%	13	13%	13	7%
>71	0	0%	6	6%	6	3%
Total	100	100%	100	100%	200	100%
Mean ±SD	7.56 ± 5.24		46.13 ± 15.49			

Among children, 68% patients were <10 years of age and 32% were > 10 years of age.

Among adults, maximum number of patients were seen in 2nd, 4th and 5th decade of life i.e. 21% each, followed by patients in 3rd (18%),6th (13%) and 7th (6%) decade of life respectively.

Mean age of incidence was 7.56 ± 5.24 years in children and 46.13 ± 15.49 years in adults.

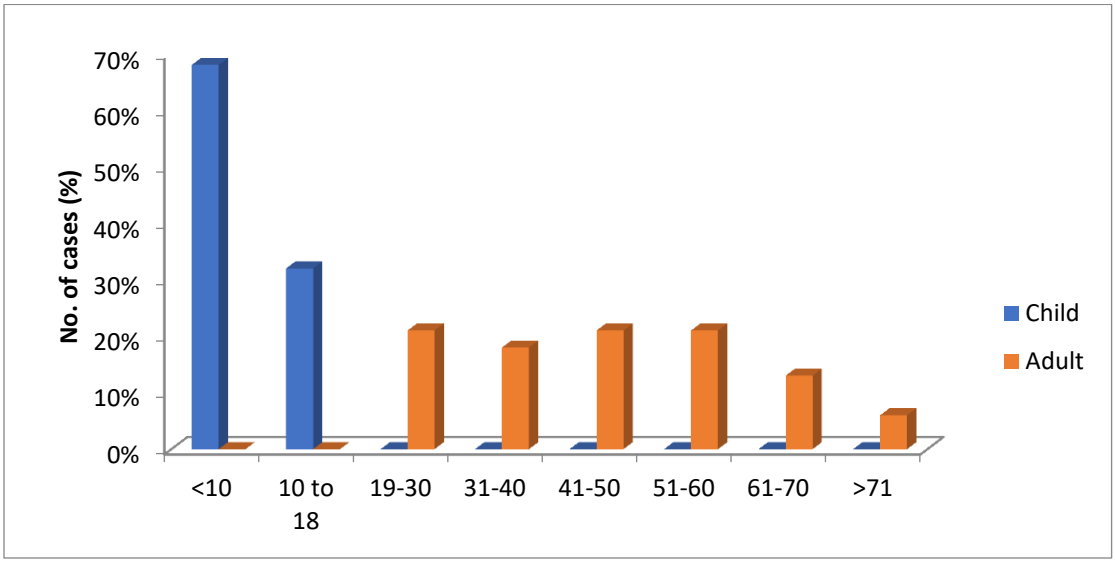


Figure 2: Age distribution of patients.

Table 3: Sex distribution of patients.

Category	Child		Adult		Total		p-value
Sex	No. of cases	Percentage	No. of cases	Percentage	No. of cases	Percentage	
Males	61	61%	56	56%	117	58%	0.473
Females	39	39%	44	44%	83	42%	

Out of total patients, 117 (58%) were males and 83 (42%) were females with a M:F ratio of 1.4:1.

In children, there were 61% males and 39% females, whereas in adults, there were 56% males and 44% females.

M:F ratio in children was 1.5:1 whereas in adults was 1.2:1.

The two groups of patients were similar in sex distribution (0.473).

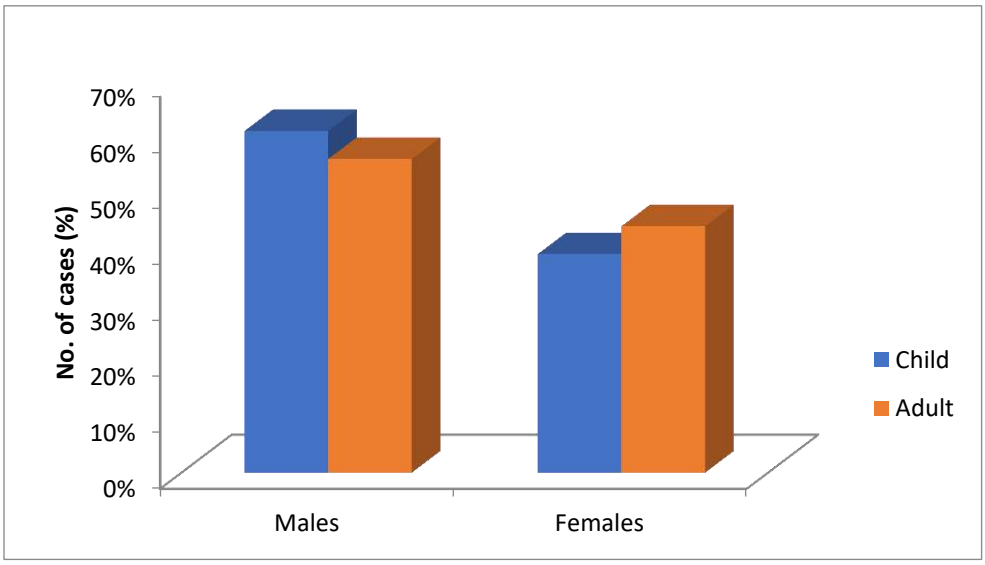


Figure 3: Sex distribution of patients.

Table 4: Distribution of patients according to site of involvement.

Category	Child		Adult		Total	
Sites involved	No. of cases	Percentage	No. of cases	Percentage	No. of cases	Percentage
Head and neck	56	56%	12	12%	68	34%
Upper limb	17	17%	6	6%	23	12%
Trunk	17	17%	42	42%	59	30%
Lower limb	10	10%	38	38%	48	24%
Genitalia	0	0%	2	2%	2	1%
Total	100	100%	100	100%	200	100%

In children, head and neck (56%) was the most common site affected, whereas trunk (42%) was commonly involved in adults, while genitalia was the least common site observed in both adult (2%) and children (0%).

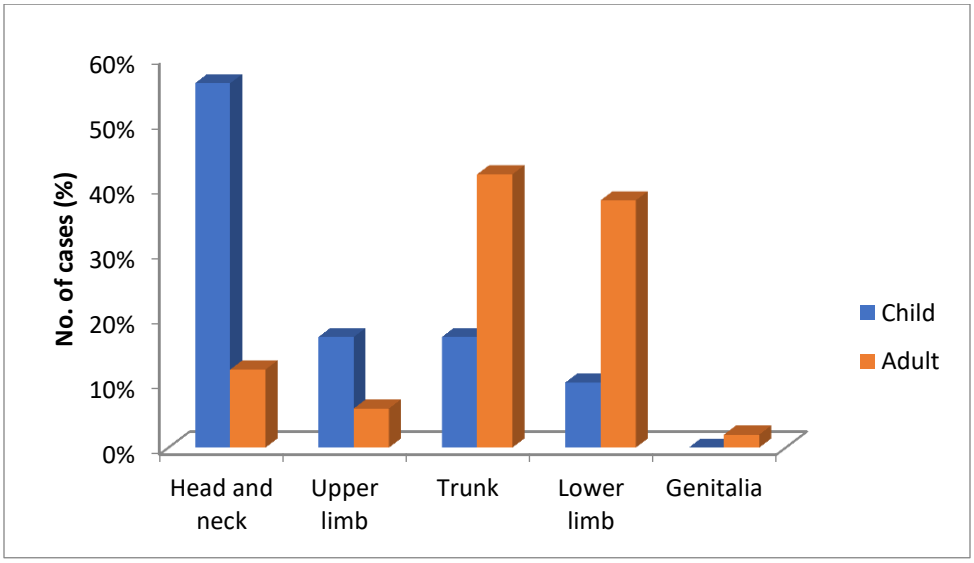


Figure 4: Distribution of patients according to site of involvement.

Table 5: Correlation of site of involvement with age in children.

Category	Age group (years)				Total	p-value
Site involved	< 10		10-18			
	No. of cases	Percentage	No. of cases	Percentage	No. of cases	
Head and neck	44	65%	12	38%	56	0.00
Upper limb	12	18%	5	16%	17	
Trunk	11	16%	6	19%	17	
Lower limb	1	1%	9	28%	10	
Total	68	100%	32	100%	100	

Head and neck was the most common site of involvement in 44 (65%) children of <10 years of age followed by upper limb in 12 (18%) cases.

The most common site of involvement in children >10years of age was head and neck in 12 (38%) cases followed by lower limb in 9 (28%) and trunk in 6 (19%) cases.

Difference in the site of involvement in two age groups is statistically significant (0.00).

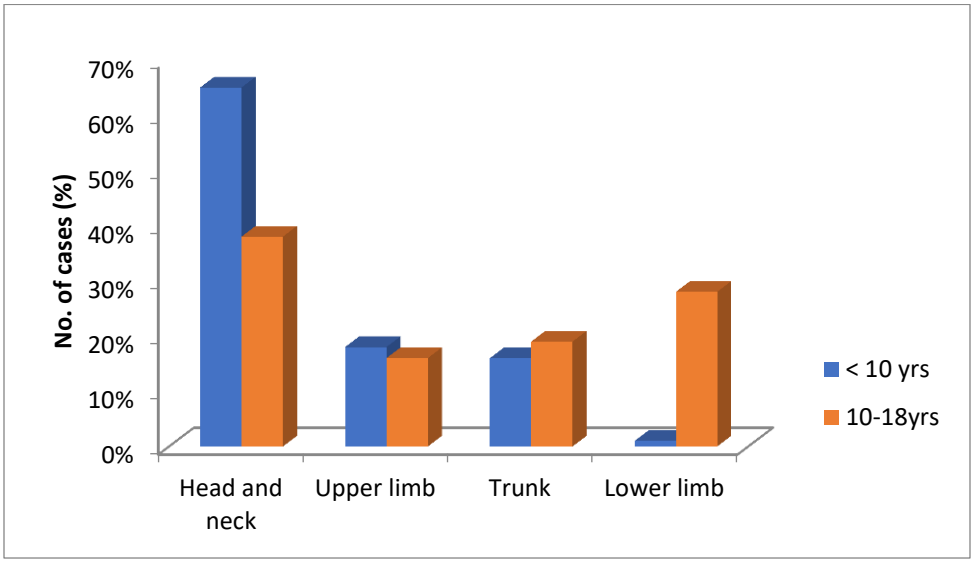


Figure 5: Correlation of site of involvement with age in children.

Table 6: Correlation of site of involvement with age in adults.

Category	Age group (years)												Total	p-value
Sites involved	< 30		31-40		41-50		51-60		61-70		> 70			
	No. of cases	%	No. of cases	%	No. of cases	%	No. of cases	%	No. of cases	%	No. of cases	%	No. of cases	
Head and neck	4	19%	2	11%	3	14%	0	0%	3	23%	0	0%	12	0.57
Upper limb	1	5%	2	11%	1	5%	1	5%	0	0%	1	17%	6	
Trunk	9	43%	9	50%	9	43%	9	43%	5	38%	1	17%	42	
Lower limb	7	33%	4	22%	8	38%	11	52%	4	31%	4	67%	38	
Genitalia	0	0%	1	6%	0	0%	0	0%	1	8%	0	0%	2	
Total	21	100%	18	100%	21	100%	21	100%	13	100%	6	100%	100	

In adults, in 2nd, 3rd, 4th, 6th decade, trunk was the most common site of involvement followed by lower limbs.

In the 5th decade the most common site of involvement was lower limbs in 11 (52%) cases followed by trunk in 9 (43%) cases.

In the 7th decade, lower limb was most common site of involvement in 4 (67%) cases followed by upper limb and trunk in 1 (17%) cases each.

However, difference in sites of involvement in adults in different decades of life was not statistically significant (0.57).

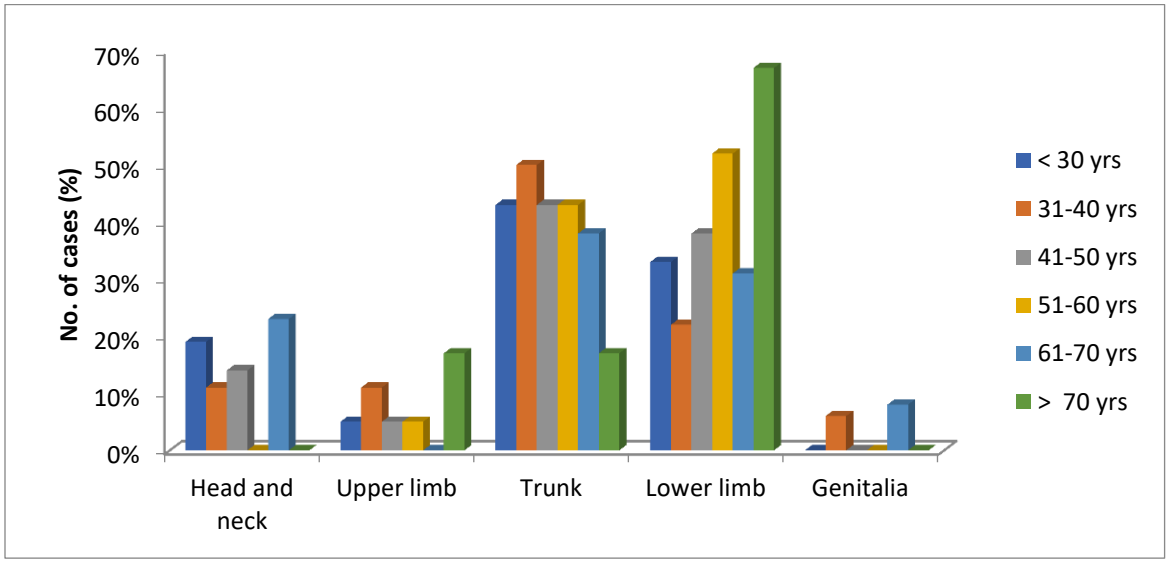


Figure 6: Correlation of site of involvement with age in adults.

ASSOCIATION WITH OTHER DISEASES/ FACTORS

Table 7: Correlation of pyodermas with other diseases/factors.

Associated diseases/ factors	Child		Adult		Total		p-value
	No. of cases	Percentage	No. of cases	Percentage	No. of cases	Percentage	
Diabetes	0	0%	31	31%	31	16%	0.00
Immunosuppression	8	8%	9	9%	17	9%	0.583
Poor hygiene	57	57%	32	32%	89	45%	0.00

Out of 16% of cases of pyoderma associated with Diabetes mellitus 31% were adults and no child with pyoderma had diabetes.

Out of 9% of cases of pyoderma associated with immunosuppression 8% were children and 9% were adults and from 45% cases of pyoderma associated with poor hygiene, 57% were children and 32% were adults.

So, significantly (0.00) more number of children with pyoderma had poor hygiene in comparison to adults.

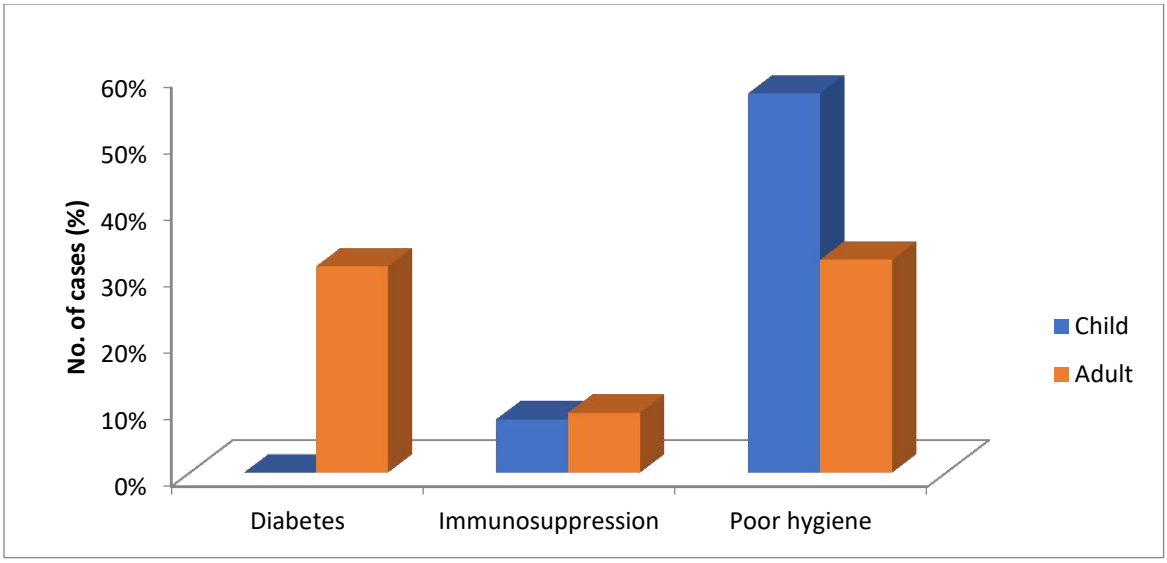


Figure 7: Correlation of pyodermas with other diseases/factors.

Table 8: Correlation of Diabetes Mellitus with Clinical diagnosis of pyoderma.

Clinical Diagnosis	Diabetes Mellitus	
	No. of cases	%
Abscess	6	19%
Infected tinea	5	16%
Folliculitis	4	13%
Furuncle	3	10%
Carbuncle	3	10%
Cellulitis	2	6%
Ecthyma	2	6%
Infected eczema	2	6%
Vasculitic ulcer	2	6%
Acute paronychia	1	3%
Sycosis barbae	1	3%
Total	31	100%

Diabetic patients most commonly presented with abscess in 6 (19%) cases followed by infected tinea in 5 (16%) cases and folliculitis in 4 (13%) cases.

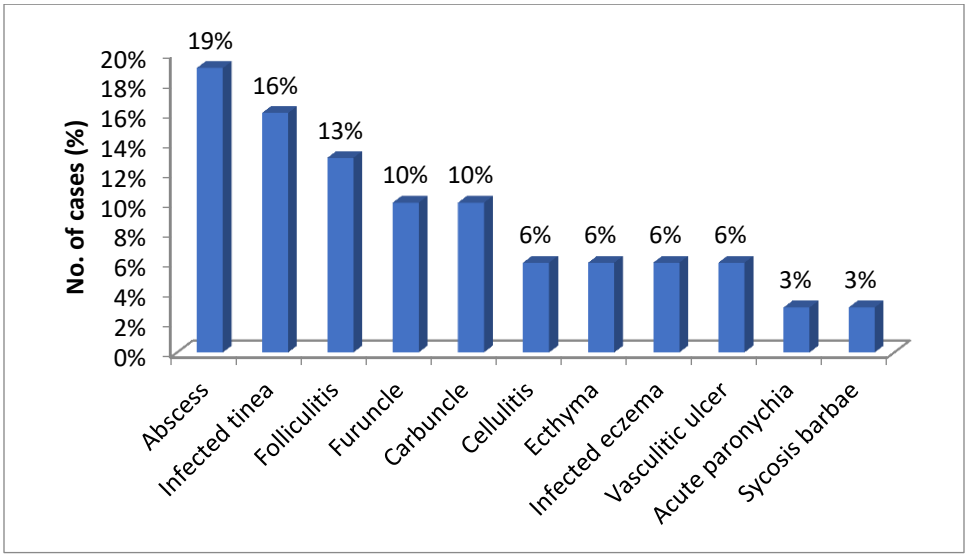


Figure 8: Correlation of Diabetes Mellitus with Clinical diagnosis of pyoderma.

Table 9: Correlation of Diabetes Mellitus with organism isolated.

Organisms isolated	Total	
	No. of cases	%
S. aureus	15	43%
P. aeruginosa	7	20%
E. coli	3	9%
K. pneumoniae	2	6%
Citrobacter	1	3%
Proteus vulgaris	1	3%
No growth	6	17%
Total	35	100%

The most common organism isolated from Diabetic patients was *S. aureus* in 15 (43%) cases followed by *P. aeruginosa* in 7 (20%) cases. *Citrobacter* and *Proteus vulgaris* were isolated in 3% cases each. No growth was found in 17% cases.

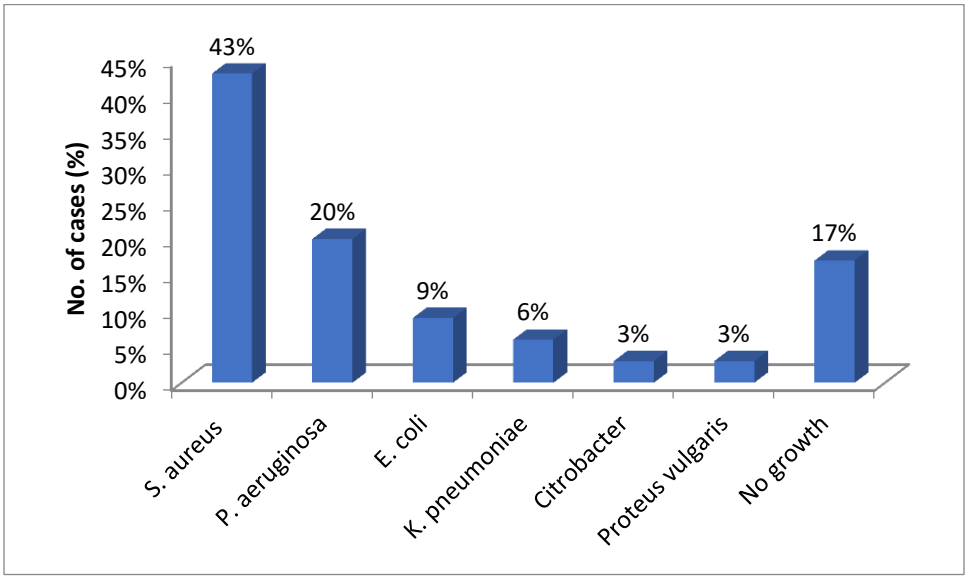


Figure 9: Correlation of Diabetes Mellitus with organism isolated.

CLINICAL DATA

Table 10: Correlation of signs and symptoms of pyodermas in children and adult.

Signs and symptoms	Yes/No	Child		Adult		Total
		No. of cases	%	No. of cases	%	No. of cases
Erythema	No	24	24%	18	18%	42
	Yes	76	76%	82	82%	158
Pain	No	20	20%	13	13%	33
	Yes	80	80%	87	87%	167
Swelling	No	89	89%	46	46%	135
	Yes	11	11%	54	54%	65
Crusting	No	30	30%	68	68%	98
	Yes	70	70%	32	32%	102
Oozing	No	72	72%	70	70%	142
	Yes	28	28%	30	30%	58

Pain (87%), erythema (82%), swelling (54%) and oozing (30%) were mostly seen in adults whereas crusting (70%) was predominantly seen in children.

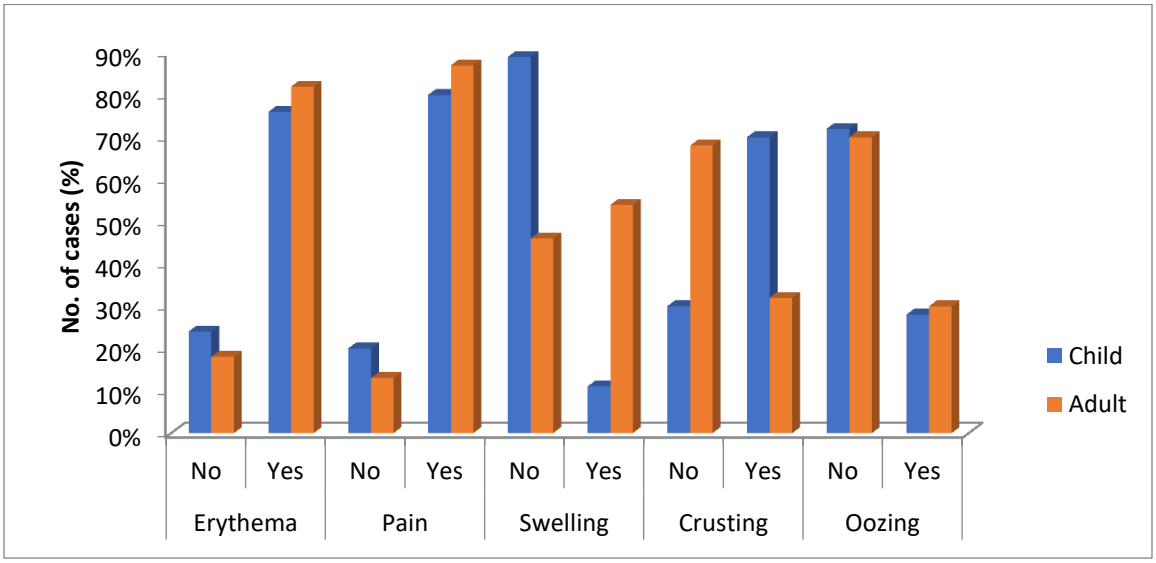


Figure 10: Correlation of signs and symptoms of pyodermas in children and adult.

Table 11: Comparison of primary and secondary pyoderma in children and adults.

Category	GROUP				Total	
Type of pyoderma	Child		Adult			
	No. of cases	%	No. of cases	%	No. of cases	%
Primary	68	47.22%	76	52.77%	144	72%
Secondary	32	57.14%	24	42.85%	56	28%

Primary pyodermas (72%) were more common than secondary pyodermas (28%).

Out of 72% patients of primary pyodermas, 68 (47.22%) were children and 76 (52.77%) were adults and from 28% cases of secondary pyodermas, 32 (57.14%) were children and 24 (42.85%) were adults.

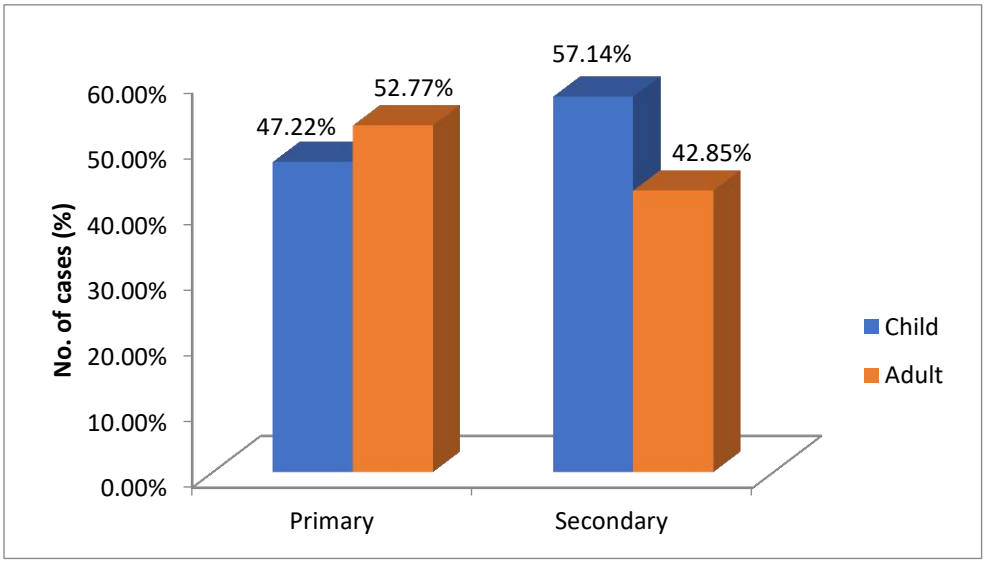


Figure 11: Comparison of primary and secondary pyoderma in children and adults.

Table 12: Comparison of primary and secondary pyoderma in children and adults visiting OPD and IPD.

Type of pyoderma	Child					Adult				
	OPD/IPD				Total	OPD/IPD				Total
	OPD		IPD			OPD		IPD		
	N0. Of cases	%	N0. Of cases	%	N0. Of cases	N0. Of cases	%	N0. Of cases	%	N0. Of cases
Primary	65	67.70%	3	75.00%	68	63	75.00%	13	81.25%	76
Secondary	31	32.30%	1	25.00%	32	21	25.00%	3	18.75%	24
Total	96	100.00%	4	100.00%	100	84	100.00%	16	100.00%	100
p-value	0.759					0.276				

Primary pyodermas were seen in 65 (67.7%) children and 63 (75%) adults visiting OPD whereas in 3 (75%) children and 13 (81.25%) adults admitted in IPD.

Secondary pyodermas were seen in 31 (32.30%) children and 21 (25%) adults visiting OPD and 1 (25%) children and 3 (18.75%) adults admitted in IPD.

However, the difference in two groups coming to either OPD or admitted in IPD was not statistically significant.

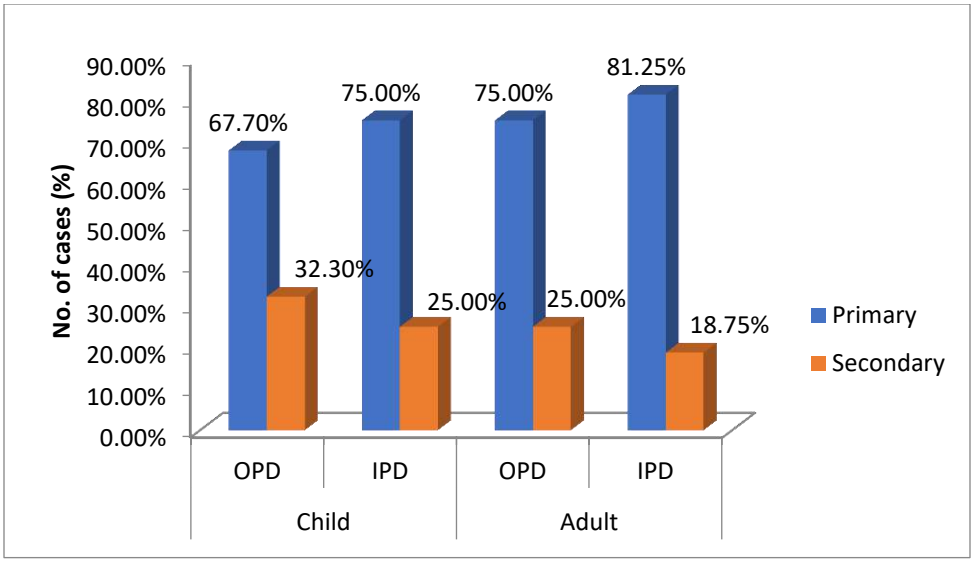


Figure 12: Comparison of primary and secondary pyoderma in children and adults visiting OPD and IPD.

Table 13: Comparison of primary and secondary pyodermas in children and adults in relation to site of involvement.

Sites involved	Child				Adult			
	Primary pyoderma		Secondary pyoderma		Primary pyoderma		Secondary pyoderma	
	No. of cases	%	No. of cases	%	No. of cases	%	No. of cases	%
Head and neck	51	75%	5	16%	12	16%	0	0%
Upper limb	3	4%	14	44%	4	5%	2	9%
Trunk	7	10%	10	31%	31	41%	11	46%
Lower limb	7	10%	3	9%	28	37%	10	41%
Genitalia	0	0%	0	0%	1	1%	1	4%
Total	68	100%	32	100%	76	100%	24	100%
p-value	0				0.28			

In primary pyoderma most common site was head and neck in 51 (75%) followed by lower limb and trunk in 7 (10%) cases each in children, whereas trunk (41%) was the most common site followed by lower limb (37%) in adults.

In secondary pyoderma, most common site was upper limb in 14 (44%) followed by trunk in 10 (31%) cases in children, whereas trunk (46%) followed by lower limb (41%) were the most common site involved in adults.

Sites of involvement were significantly different for primary and secondary pyodermas in children (0.00).

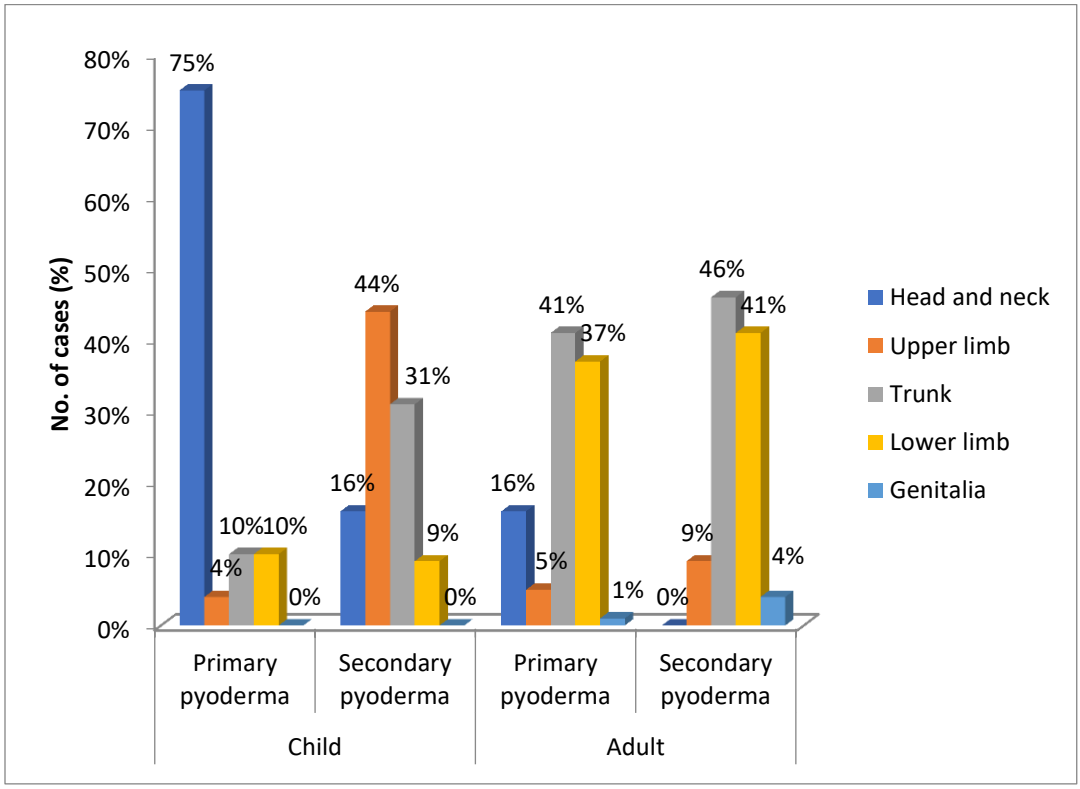


Figure 13: Comparison of primary and secondary pyodermas in children and adults in relation to site of involvement.

Table 14: Comparison of distribution of clinical sub-types of primary pyoderma in children and adults.

Clinical sub-types of Primary pyoderma	Child		Adult		Total	
	No. of cases	%	No. of cases	%	No. of cases	%
Impetigo contagiosa	43	63%	0	0%	43	30%
Folliculitis	12	18%	22	29%	34	24%
Furuncle	9	13%	14	18%	23	16%
Abscess	2	3%	17	22%	19	13%
Cellulitis	1	1%	6	8%	7	5%
Ecthyma	0	0%	7	9%	7	5%
Carbuncle	1	1%	5	7%	6	3%
DCPA	0	0%	2	3%	2	1.50%
Sycosis barbae	0	0%	2	3%	2	1.50%
Acute paronychia	0	0%	1	1%	1	1.00%
Total	68	100%	76	100%	144	100%

The most common clinical diagnosis among primary pyodermas was Impetigo contagiosa (63%) which was seen only in pediatric age group. Folliculitis (29%) was most common in adults, followed by abscess (22%) and furuncle (18%) in decreasing order of frequency.

Ecthyma (9%), DCPA (3%), sycosis barbae (3%), acute paronychia (1%) were seen only in adults.

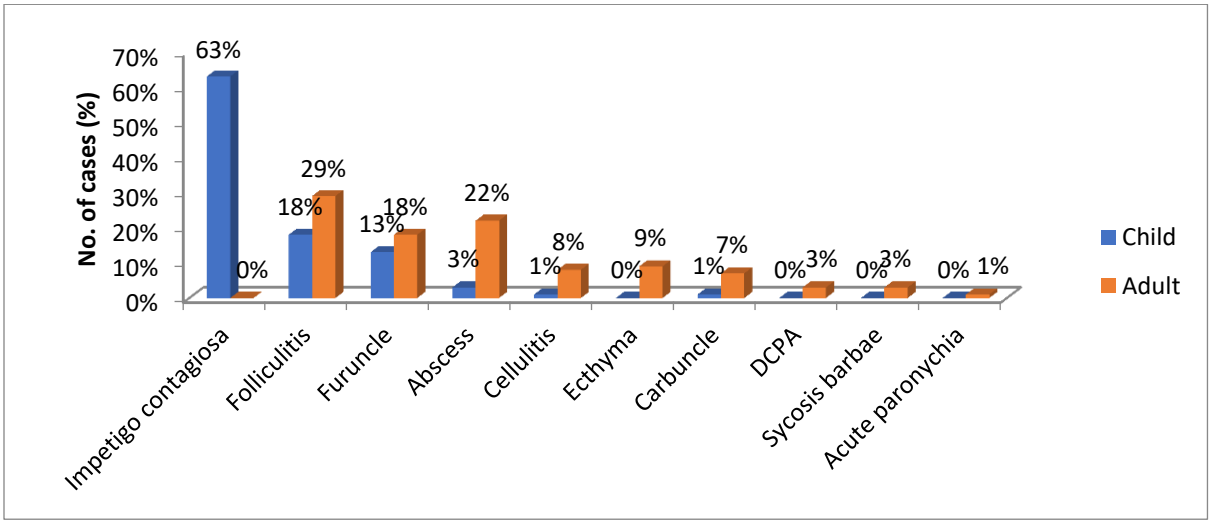


Figure 14: Comparison of distribution of clinical sub-types of primary pyoderma in children and adults.

Table 15: Comparison of distribution of clinical sub-types of primary pyoderma in children and adults visiting OPD and IPD.

Clinical sub-types of **Primary pyoderma**	Child					Adult				
	OPD/IPD				Total	OPD/IPD				Total
	OPD		IPD			OPD		IPD		
	No. of cases	%	No. of cases	%	No. of cases	No. of cases	%	No. of cases	%	No. of cases
Impetigo contagiosa	42	64.6%	1	33.3%	43	0	0.0%	0	0.0%	0
Folliculitis	12	18.5%	0	0.0%	12	22	34.9%	0	0.0%	22
Furuncle	7	10.8%	2	66.7%	9	11	17.5%	3	21.4%	14
Abscess	2	3.1%	0	0.0%	2	14	22.2%	3	21.4%	17
Cellulitis	1	1.5%	0	0.0%	1	3	4.8%	3	21.4%	6
Ecthyma	0	0.0%	0	0.0%	0	5	7.9%	2	14.3%	7
Carbuncle	1	1.5%	0	0.0%	1	2	3.2%	3	21.4%	5
DCPA	0	0.0%	0	0.0%	0	2	3.2%	0	0.0%	2
Sycosis barbae	0	0.0%	0	0.0%	0	2	3.2%	0	0.0%	2
Acute paronychia	0	0.0%	0	0.0%	0	1	1.6%	0	0.0%	1
Total	65	100.0%	3	100.0%	68	62	100.0%	14	100.0%	76
p-value	0.159					0.052				

The most common clinical diagnosis among primary pyoderma in children was impetigo contagiosa (64.6%) followed by folliculitis (18.5%) visiting OPD, and in IPD furuncle (66.7%) was most commonly seen followed by impetigo contagiosa (33.3%).

The most common clinical diagnosis among primary pyoderma in adults visiting OPD was folliculitis (34.9%) followed by abscess (22.2%) and in IPD furuncle (21.4%), abscess (21.4%), cellulitis (21.4%), carbuncle (21.4%) were more commonly seen.

So, difference in pattern of primary pyodermas in OPD and IPD patients was not statistically significant in both the groups.

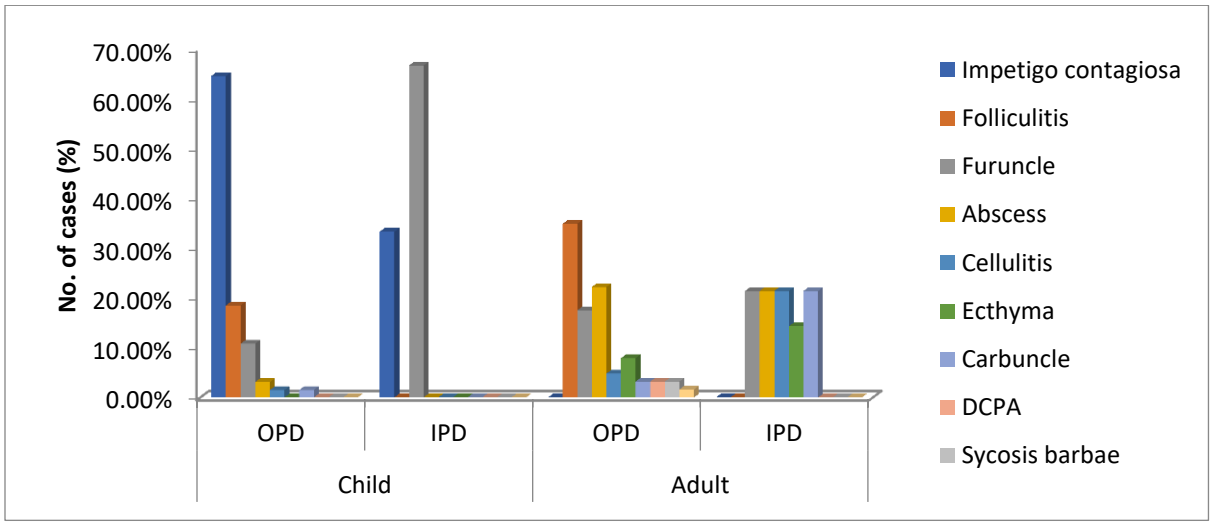


Figure 15: Comparison of distribution of clinical sub-types of primary pyoderma in children and adults visiting OPD and IPD.

Table 16: Comparison of distribution of clinical sub-types of secondary pyodermas in children and adults.

Clinical sub-types of Secondary pyoderma	Child		Adult		Total	
	No. of cases	%	No. of cases	%	No. of cases	%
Infected tinea	4	13%	13	57%	17	30%
Infected scabies	15	47%	2	4%	17	30%
Infected eczema	9	28%	5	22%	14	25%
Infected EB	1	3%	0	0%	1	2%
Infected hemangioma	1	3%	0	0%	1	2%
Infected LP	0	0%	1	4%	1	2%
Infected LSC	0	0%	1	4%	1	2%
Infected pediculosis	1	3%	0	0%	1	2%
Infected umbilical stump	1	3%	0	0%	1	2%
Vasculitic ulcer	0	0%	2	9%	2	3%
Total	32	100%	24	100%	56	100%

The most common clinical diagnosis among secondary pyoderma was infected tinea in 57% adults, whereas infected scabies (47%) was most commonly seen in children.

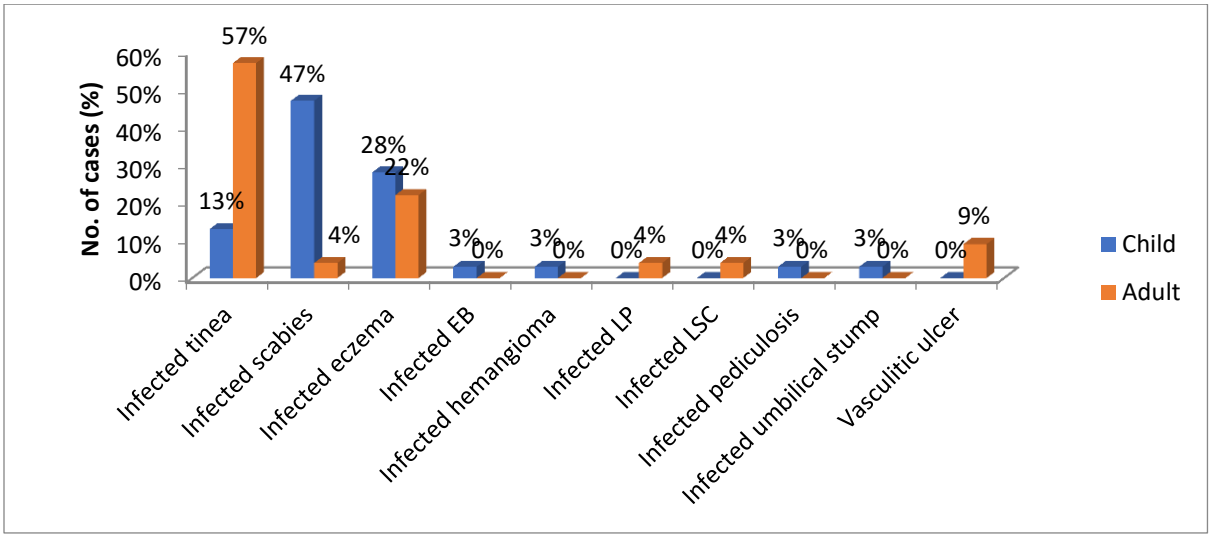


Figure 16: Comparison of distribution of clinical sub-types of secondary pyodermas in children and adults.

Table 17: Comparison of distribution of clinical sub-types of secondary pyodermas in children and adults visiting OPD and IPD.

Clinical sub-types of **Secondary pyoderma**	Child					Adult				
	OPD/IPD				Total	OPD/IPD				Total
	OPD		IPD			OPD		IPD		
	No. of cases	%	No. of cases	%	No. of cases	No. of cases	%	No. of cases	%	No. of cases
Infected scabies	15	48.4%	0	0.0%	15	2	9%	0	0%	2
Infected eczema	8	25.8%	1	100.0%	9	5	23%	0	0.0%	5
Infected tinea	4	12.9%	0	0.0%	4	12	54.5%	1	50%	13
Infected hemangioma	1	3.2%	0	0.0%	1	0	0.0%	0	0.0%	0
Infected pediculosis	1	3.2%	0	0.0%	1	0	0.0%	0	0.0%	0
Infected EB	1	3.2%	0	0.0%	1	0	0.0%	0	0.0%	0
Infected umbilical stump	1	3.2%	0	0.0%	1	0	0.0%	0	0.0%	0
Vasculitic ulcer	0	0.0%	0	0.0%	0	1	4.5%	1	50%	2
Infected LP	0	0.0%	0	0.0%	0	1	4.5%	0	0.0%	1
Infected LSC	0	0.0%	0	0.0%	0	1	4.5%	0	0.0%	1
Total	31	100.0%	1	100.0%	32	22	100.0%	2	100.0%	24
p-value	0.853					0.407				

The most common clinical diagnosis among secondary pyoderma in children was infected scabies (48.4%) followed by infected eczema (25.8%) visiting OPD, and in IPD infected eczema (100%) was exclusively seen.

The most common clinical diagnosis among secondary pyoderma in adults was infected tinea (54.5%) followed by infected eczema (23%) visiting OPD and in IPD, only infected tinea (50%) and vasculitic ulcer (50%) were seen.

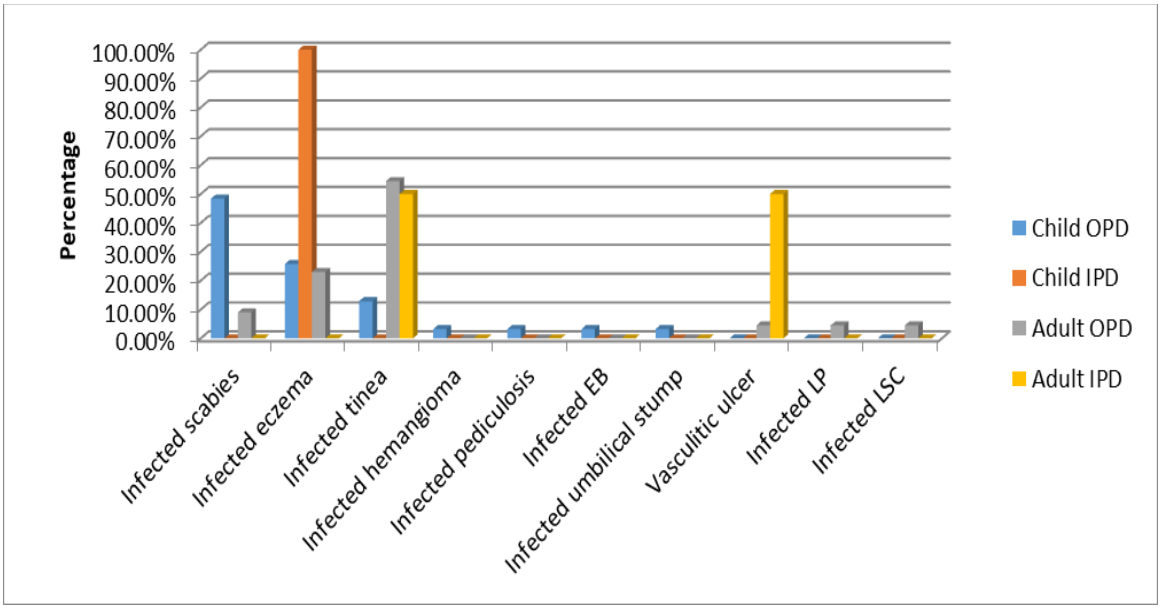


Figure 17: Comparison of distribution of clinical sub-types of secondary pyodermas in children and adults visiting OPD and IPD.

Table 18: Correlation of clinical diagnosis of pyodermas with age in children.

Clinical Diagnosis of pyodermas in **Children**	Age group (years)				Total	
	<10		10-18			
	No. of cases	%	No. of cases	%	No. of cases	%
Impetigo contagiosa	38	56%	5	16%	43	43%
Folliculitis	6	8%	6	18%	12	12%
Furuncle	2	2%	7	22%	9	9%
Abscess	0	0%	2	6%	2	2%
Carbuncle	0	0%	1	3%	1	1%
Cellulitis	0	0%	1	3%	1	1%
Infected scabies	12	18%	3	10%	15	15%
Infected eczema	7	10%	2	6%	9	9%
Infected tinea	1	2%	3	10%	4	4%
Infected hemangioma	1	2%	0	0%	1	1%
Infected pediculosis	0	0%	1	3%	1	1%
Infected EB	0	0%	1	3%	1	1%
Infected umbilical stump	1	2%	0	0%	1	1
Total	68	100%	32	100%	100	100%
p-value	0.00					

In <10years age group, impetigo contagiosa (56%) was most commonly seen followed by folliculitis (8%) among primary pyoderma and in secondary pyoderma infected scabies (18%) was most common followed by infected eczema (10%).

In 1st decade, most common clinical diagnosis was furuncle (22%) followed by folliculitis (18%) among primary pyodermas, whereas in secondary pyodermas, infected scabies (10%) and infected tinea (10%) were most commonly seen.

Statistically significant (0.00) difference were seen in types of pyoderma in different age groups in children.

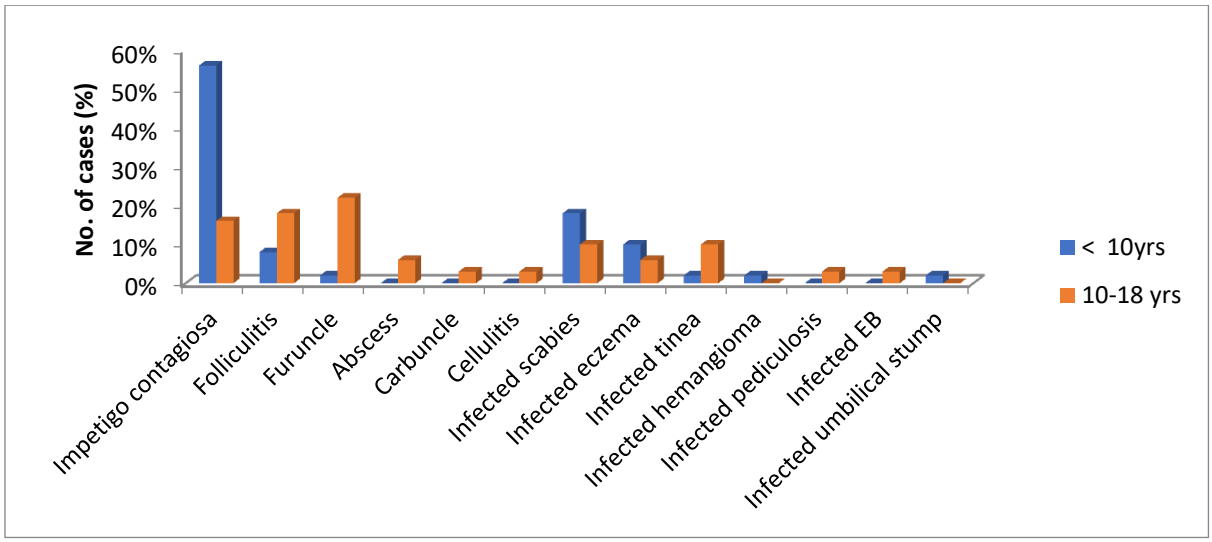


Figure 18: Correlation of clinical diagnosis of pyodermas with age in children.

Table 19: Correlation of clinical diagnosis of pyodermas with age in adults.

Clinical Diagnosis of pyodermas in Adults	Age group (years)													
	< 30		31-40		41-50		51-60		61-70		> 70		Total	
	No. of cases	%	No. of cases	%	No. of cases	%	No. of cases	%	No. of cases	%	No. of cases	%	No. of cases	%
Folliculitis	8	38%	4	22%	2	9%	4	20%	3	23%	1	16%	22	22%
Abscess	4	20%	4	22%	5	23%	3	14%	1	7%	0	0%	17	17%
Furuncle	4	20%	3	16%	2	9%	2	9%	2	15%	1	16%	14	14%
Ecthyma	0	0%	2	12%	1	4%	1	4%	2	15%	1	16%	7	7%
Cellulitis	0	0%	0	0%	1	4%	3	14%	1	7%	1	16%	6	6%
Carbuncle	0	0%	1	5%	1	4%	2	9%	1	7%	0	0%	5	5%
DCPA	0	0%	1	5%	0	0%	1	4%	0	0%	0	0%	2	2%
Acute paronychia	0	0%	1	5%	0	0%	0	0%	0	0%	0	0%	1	1%
Infected tinea	1	4%	2	12%	5	23%	3	14%	2	15%	0	0%	13	13%
Infected eczema	3	14%	0	0%	0	0%	1	4%	0	0%	1	16%	5	5%
Infected scabies	0	0%	0	0%	0	0%	1	4%	1	7%	0	0%	2	2%
Sycosis barbae	0	0%	0	0%	2	9%	0	0%	0	0%	0	0%	2	2%
Vasculitic ulcer	0	0%	0	0%	1	4%	0	0%	0	0%	1	16%	2	2%
Infected LP	0	0%	0	0%	1	4%	0	0%	0	0%	0	0%	1	1%
Infected LSC	1	4%	0	0%	0	0%	0	0%	0	0%	0	0%	1	1%
Total	21	21%	18	18%	21	21%	21	21%	13	13%	6	6%	100	100%
p-value	0.542													

In the 2nd decade, the most common diagnosis was folliculitis (38%) followed by abscess (20%) and furuncle (20%).

In the 3rd decade, most common diagnosis were folliculitis (22%) and abscess (22%)

In the 4th decade, abscess (23%) and infected tinea (23%) were commonly seen.

In the 5th decade, folliculitis (20%), abscess (14%), cellulitis (14%) and infected tinea (14%) were seen.

In 6th decade, folliculitis (23%) was most commonly seen followed by ecthyma (15%) and furuncle (15%).

In 7th decade, folliculitis, cellulitis, ecthyma, furuncle, infected eczema and vasculitic ulcer were seen in 16% each.

Differences in types of pyoderma in different decades of life in adults were not significant.

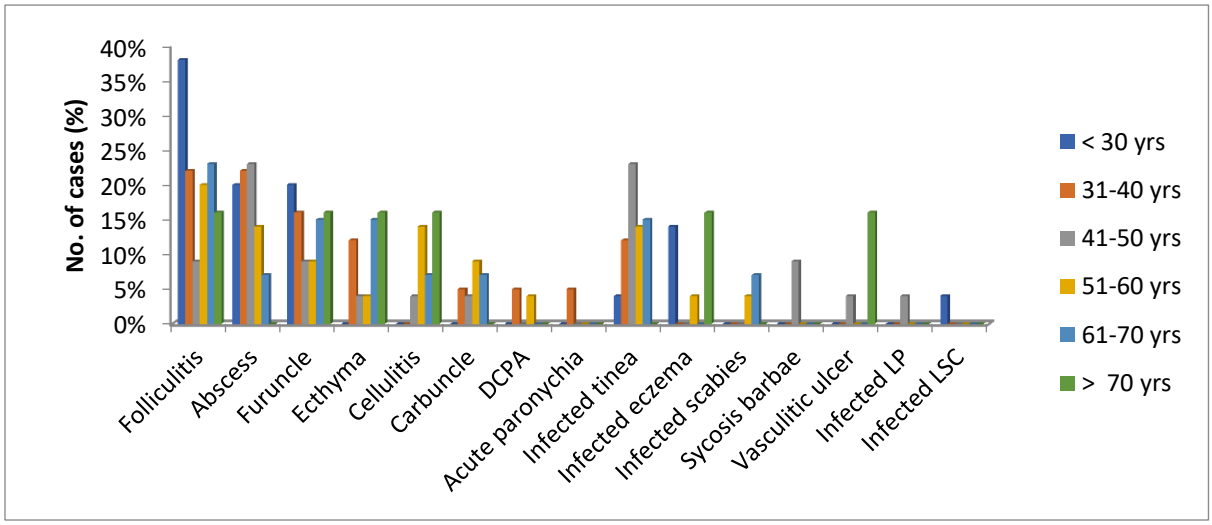


Figure 19: Correlation of clinical diagnosis of pyodermas with age in adults.

Table 20: Correlation of site of involvement with clinical diagnosis in children and adult.

Sites involved	Clinical diagnosis	Child		Adult		Total
		No. of cases	%	No. of cases	%	
Head and neck	Impetigo contagiosa	41	73%	0	0%	41
	Folliculitis	9	16%	7	58%	16
	Furuncle	0	0%	2	17%	2
	Cellulitis	1	2%	0	0%	1
	Ecthyma	0	0%	1	8%	1
	Infected eczema	3	5%	0	0%	3
	Sycosis barbae	0	0%	2	17%	2
	Infected pediculosis	1	2%	0	0%	1
	Infected tinea	1	2%	0	0%	1
	Total	56	100%	12	100%	68
Upper limb	Furuncle	1	6%	2	33%	3
	Impetigo contagiosa	2	12%	0	0%	2
	Folliculitis	0	0%	1	17%	1
	Abscess	0	0%	1	17%	1
	Infected scabies	13	76%	0	0%	13
	Infected eczema	1	6%	1	17%	2
	Vasculitic ulcer	0	0%	1	17%	1
	Total	17	100%	6	100%	23
Trunk	Abscess	1	6%	14	33%	15
	Furuncle	3	18%	7	17%	10
	Folliculitis	3	18%	6	14%	9
	Carbuncle	0	0%	4	10%	4
	Infected tinea	3	18%	8	19%	11
	Infected eczema	5	29%	1	2%	6
	Infected scabies	1	6%	2	5%	3

	Infected umbilical stump	1	6%	0	0%	1
	Total	17	100%	42	100%	59
Lower limb	Furuncle	5	50%	3	8%	8
	Folliculitis	0	0%	7	18%	7
	Ecthyma	0	0%	6	16%	6
	Cellulitis	0	0%	6	16%	6
	Abscess	1	10%	2	5%	3
	Carbuncle	1	10%	1	3%	2
	DCPA	0	0%	2	5%	2
	Acute paronychia	0	0%	1	3%	1
	Infected tinea	0	0%	4	11%	4
	Infected eczema	0	0%	3	8%	3
	Infected EB	1	10%	0	0%	1
	Infected hemangioma	1	10%	0	0%	1
	Infected LP	0	0%	1	3%	1
	Infected LSC	0	0%	1	3%	1
	Infected scabies	1	10%	0	0%	1
	Vasculitic ulcer	0	0%	1	3%	1
	Total	10	100%	38	100%	48
Genitalia	Folliculitis	0	0%	1	50%	1
	Infected tinea	0	0%	1	50%	1
	Total	0	0%	2	100%	2

Over head and neck, impetigo contagiosa was most commonly seen in children (73%) whereas in adults, folliculitis (58%) was seen.

Over upper limb, infected scabies was seen in children (76%) and furuncle in adults (33%).

Over trunk, infected eczema was seen in children (29%) whereas abscess (33%) in adults.

Over lower limbs, furuncle was seen in children (50%) whereas in adults, folliculitis (18%) was seen.

Folliculitis and infected tinea were seen in one adult patient each (50%), however, no patient in pediatric age group had pyoderma in the genital area.

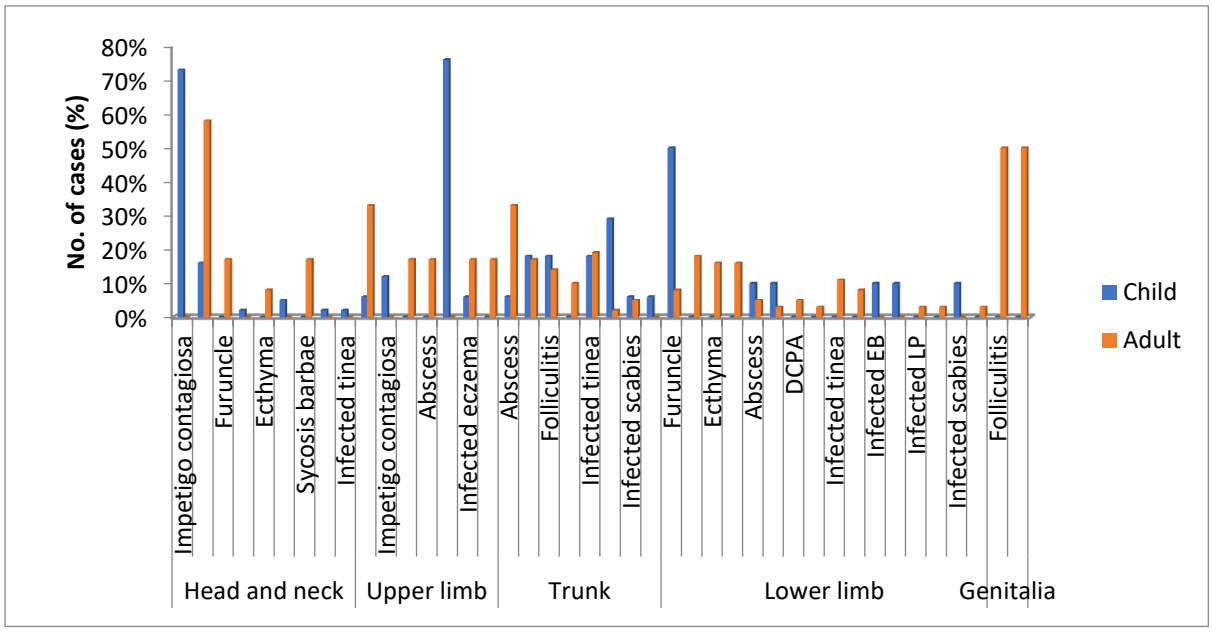


Figure 20: Correlation of site of involvement with clinical diagnosis in children and adult.

LABORATORY DATA

Table 21: Type of organism seen in pus samples of patients visiting OPD and IPD.

Gram's staining	Child				Adult				Total	
	OPD		IPD		OPD		IPD			
	No. of samples	%	No. of samples	%	No. of samples	%	No. of samples	%	No. of samples	%
Gram Positive	88	61%	1	1%	51	36%	4	2%	144	72%
Gram Negative	3	12%	1	4%	11	45%	9	39%	24	12%
Both Gram Positive +Gram Negative	1	17%	0	0%	3	50%	2	33%	6	4%
No organism	4	15%	2	7%	19	74%	1	4%	26	14%
Total	96	48%	4	2%	84	42%	16	8%	200	100%

Out of total 200 pus samples, 144 (72%) had gram positive cocci and 24 (12%) showed gram negative bacilli while other 6 (4%) showed both gram positive and negative organisms. No organism was seen in 26 (14%) samples.

Gram positive cocci was seen in 88 (61%) children and 51 (36%) adults in OPD samples whereas in IPD samples in 1 (1%) child and in 4 (2%) adults.

Gram negative bacilli was seen in 3 (12%) children and 11 (45%) adults in OPD samples whereas in IPD samples in 1 (4%) child and 9 (39%) adults.

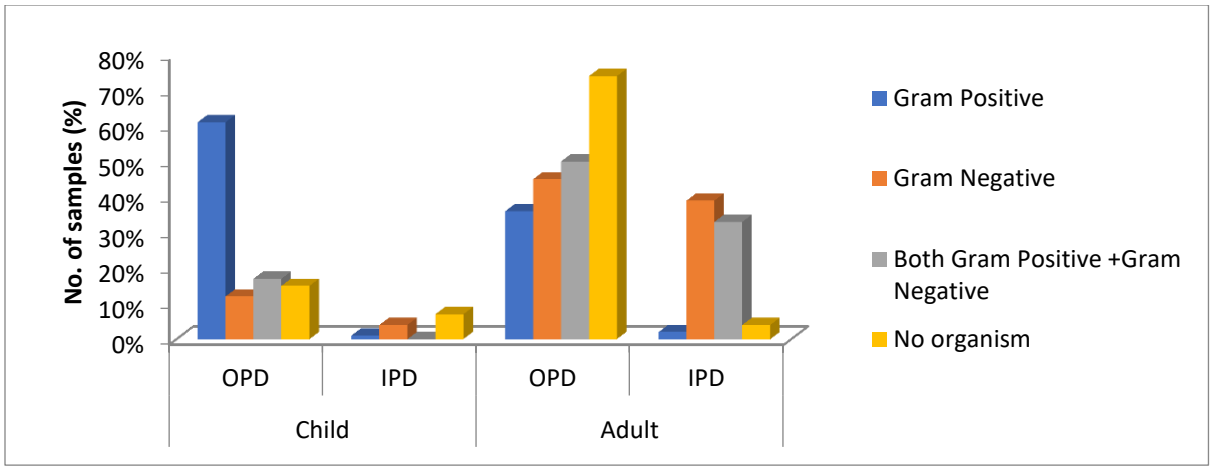


Figure 21: Type of organism seen in pus samples of patients visiting OPD and IPD.

Table 22: Number of organisms isolated per pus sample.

No. of Organisms isolated per pus sample	GROUP				Total	
	Child		Adult			
	No. of samples	%	No. of samples	%	No. of samples	%
Monomicrobial	92	92%	70	70%	162	82%
Polymicrobial	2	2%	10	10%	12	5%
No organism	6	6%	20	20%	26	13%

In 162 (82%) pus samples, monomicrobial growth was obtained, polymicrobial growth (isolated two organisms per sample) was seen in 12 (5%) pus samples.

Monomicrobial growth was seen in 92% children and 70% adults, whereas polymicrobial growth was in 2% children and 10% adults.

There were total 186 organisms isolated in 174 pus samples.

No organism (13%) was seen in 6% children and 20% adults.

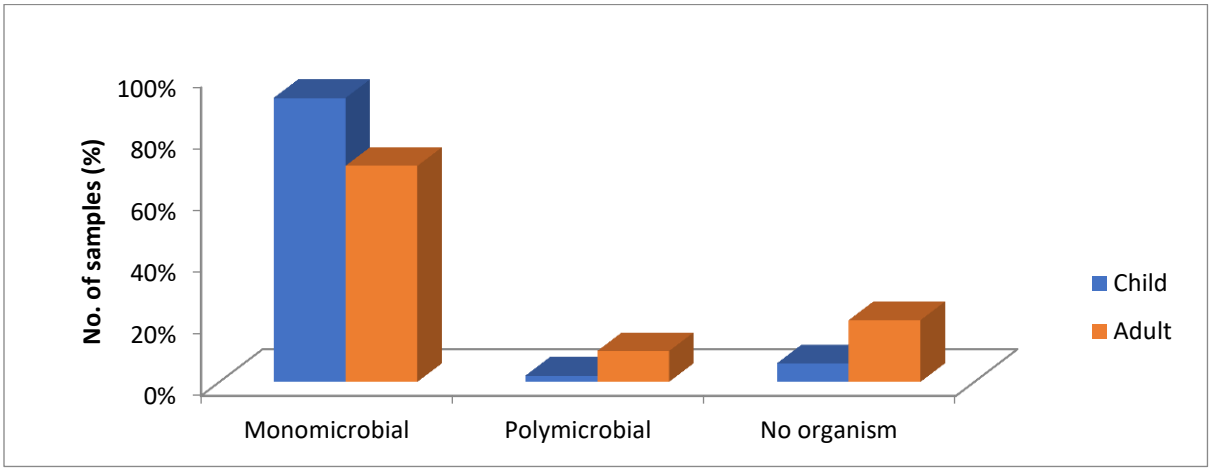


Figure 22: Number of organisms isolated per pus sample.

Table 23: Comparison of organisms isolated in pus samples of pyodermas in children and adults.

Gram's staining	**organism isolated**	Child		Adult		Total		
		No. of cases	%	No. of cases	%	No. of cases	%	No. of cases (%)
Gram positive organism	*S. aureus*	88	92%	58	65%	146	77%	80% (151)
	Enterococcus	2	2%	1	1%	3	2%	
	β-haemolytic streptococci	1	1%	1	1%	2	1%	
Gram negative organism	*E. coli*	4	4%	9	10%	13	7%	20% (35)
	P. aeruginosa	1	1%	12	14%	13	7%	
	K. pneumoniae	0	0%	2	2%	2	1%	
	Enterobacter	0	0%	2	2%	2	1%	
	Citrobacter	0	0%	2	2%	2	1%	
	Proteus mirabilis	0	0%	1	1%	1	1%	
	Proteus vulgaris	0	0%	1	1%	1	1%	
	Acinetobacter baumannii	0	0%	1	1%	1	1%	
	Total	96	100%	90	100%	186	100%	

S. aureus was seen in 92% children and 65% adults.

K. pneumoniae (2%), Enterobacter (2%), *Citrobacter* (2%), *Proteus mirabilis* (1%), *Proteus vulgaris* (1%), *Acinetobacter baumannii* (1%) were exclusively seen in adults.

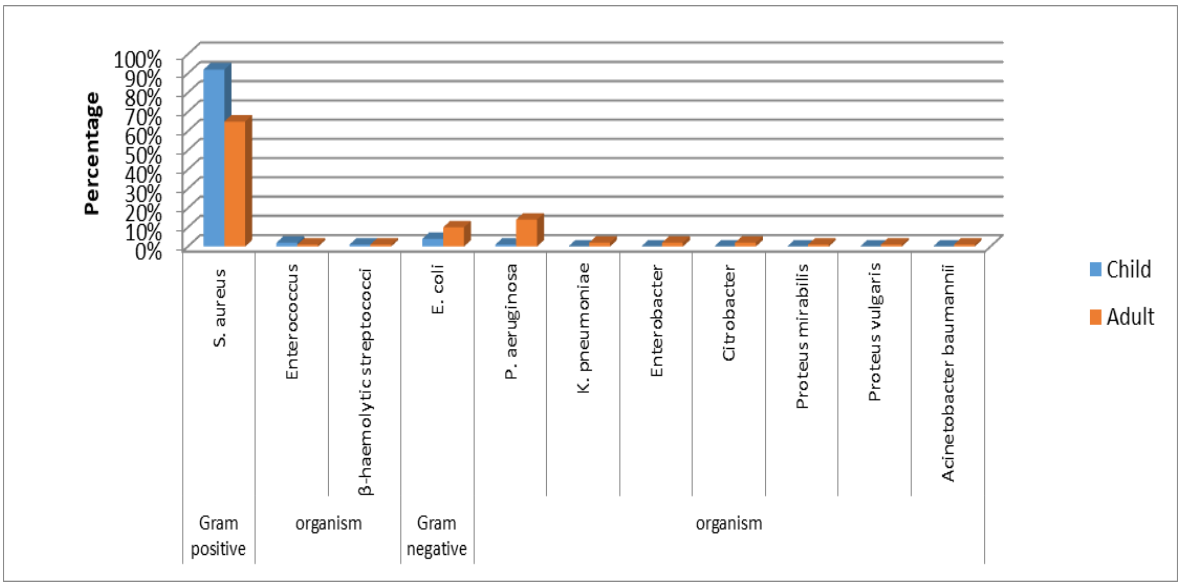


Figure 23: Comparison of organisms isolated in pus samples of pyodermas in children and adults.

Table 24: Comparison of organism isolated in OPD and IPD cases.

Organism isolated	OPD		IPD	
	No. of cases	%	No. of cases	%
S. aureus	139	85%	7	33%
Enterococcus	3	2%	0	0%
β-haemolytic streptococci	2	1%	0	0%
E. coli	11	6%	2	10%
P. aeruginosa	8	5%	5	22%
Citrobacter	1	0.5%	1	5%
Enterobacter	1	0.5%	1	5%
K. pneumoniae	0	0%	2	10%
Acinetobacter baumannii	0	0%	1	5%
Proteus mirabilis	0	0%	1	5%
Proteus vulgaris	0	0%	1	5%
total	165	100%	21	100%

S. aureus was most commonly isolated in OPD and IPD i.e. 139 (85%) OPD cases and 7 (33%) IPD cases.

E. coli was isolated in 11 (6%) OPD and 2 (10%) IPD cases.

Enterococcus (2%), β-haemolytic streptococci (1%) were seen only in OPD patients.

K. pneumoniae (10%), *Acinetobacter baumannii* (5%), *Proteus mirabilis* (5%), *Proteus vulgaris* (5%) were seen only in IPD patients.

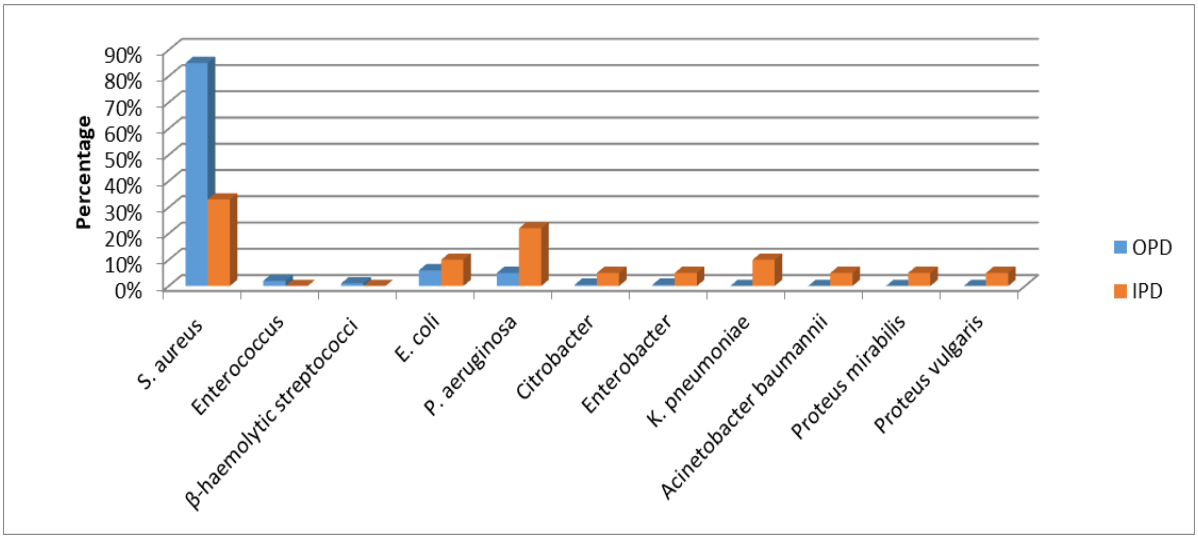


Figure 24: Comparison of organism isolated in OPD and IPD cases.

Table 25: Comparison of organism isolated in child and adults visiting OPD and IPD.

Organism isolated	Child					Adult				
	OPD/IPD				Total	OPD/IPD				Total
	OPD		IPD			OPD		IPD		
	No. of cases	%	No. of cases	%	No. of cases	No. of cases	%	No. of cases	%	No. of cases
S. aureus	87	88.80%	1	25.00%	88	52	57.80%	6	30.00%	58
P. aeruginosa	0	0.00%	1	25.00%	1	8	8.90%	4	20.00%	12
E. coli	4	4.10%	0	0.00%	4	7	7.80%	2	10.00%	9
Citrobacter	0	0.00%	0	0.00%	0	1	1.10%	1	5.00%	2
Enterobacter	0	0.00%	0	0.00%	0	1	1.10%	1	5.00%	2
K. pneumoniae	0	0.00%	0	0.00%	0	0	0.00%	2	10.00%	2
β-haemolytic streptococci	1	1.00%	0	0.00%	1	1	1.10%	0	0.00%	1
Enterococcus	2	2.00%	0	0.00%	2	1	1.10%	0	0.00%	1
Acinetobacter baumannii	0	0.00%	0	0.00%	0	0	0.00%	1	5.00%	1
Proteus mirabilis	0	0.00%	0	0.00%	0	0	0.00%	1	5.00%	1
Proteus vulgaris	0	0.00%	0	0.00%	0	0	0.00%	1	5.00%	1
No growth	4	4.10%	2	50.00%	6	19	21.10%	1	5.00%	20
Total	98	100.00%	4	100.00%	102	90	100.00%	20	100.00%	110
p-value	0.001					0.001				

S. aureus was most commonly seen organism in OPD as compared to IPD patients. It was isolated in 87 (88.8%) children and 52 (57.8%) adults in OPD whereas in 1 (25%) child and 6 (30%) adults in IPD.

E. coli was seen in OPD patients, 4 (4.1%) children and 7 (7.8%) adults whereas in 2 (10%) IPD adults.

P. aeruginosa was found in 8 (8.90%) adults in OPD whereas in 1 (25%) child and 4 (20%) adults in IPD.

β-haemolytic streptococci was seen only in OPD patients in 1 (1%) child and 1 (1.10%) adult.

Enterococcus was seen only in OPD patients in 2% children and 1.10% adults.

Proteus mirabilis (5%), *Proteus vulgaris* (5%), *Acinetobacter baumannii* (5%), were exclusively seen in IPD adults.

Citrobacter and *Enterobacter* were isolated in adults in 1.10% OPD and 5% IPD cases.

K. pneumoniae was exclusively isolated in 10% IPD adults.

No growth was obtained in 4.10% children and 21.10% adults in OPD whereas in 50% children and 5% adults in IPD, there was no growth on culture.

So, bacteria isolated from OPD and IPD differed significantly both in adults (0.001) and children (0.001).

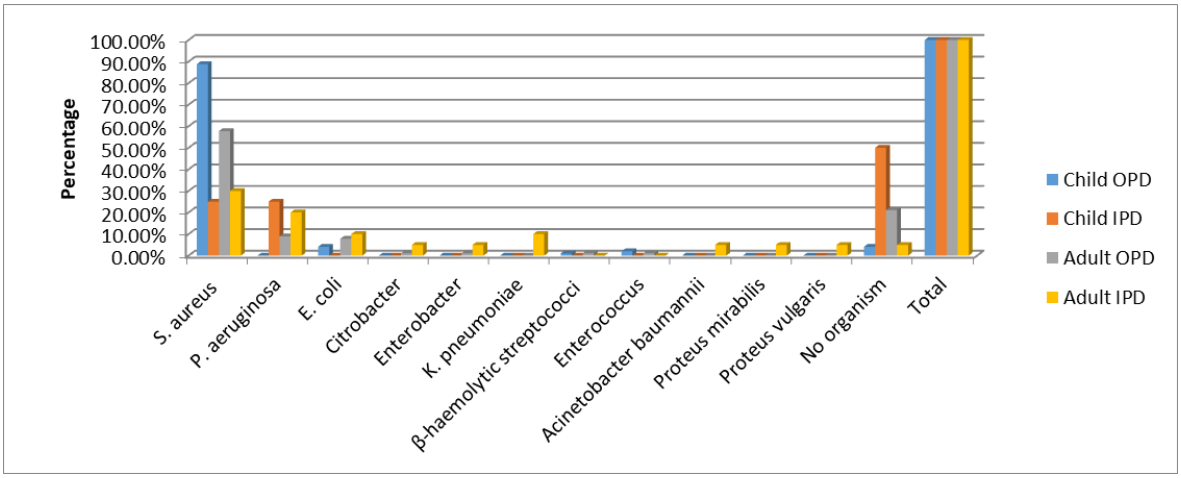


Figure 25: Comparison of organism isolated in child and adults visiting OPD and IPD.

Table 26: Comparison of MRSA isolated in child and adults.

Organism isolated	Child		Adult		Total	
	No. of cases	%	No. of cases	%	No. of cases	%
MSSA	85	97%	55	95%	140	96%
MRSA	3	3%	3	5%	6	4%
Total	88	100%	58	100%	146	100%

MRSA was obtained in pus samples of 3% children and 5% adults respectively.

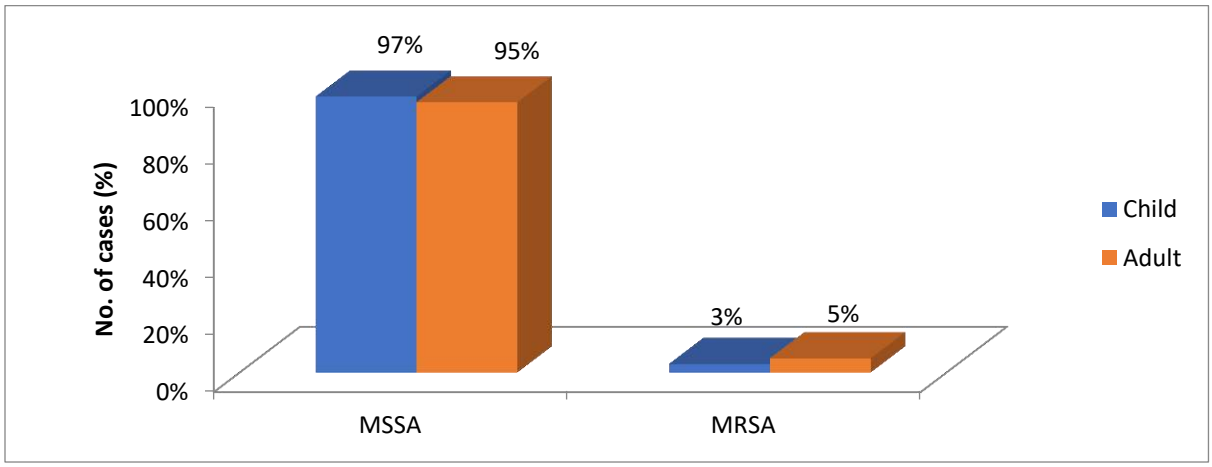


Figure 26: Comparison of MRSA isolated in child and adults.

Table 27: Comparison of MRSA isolated in child and adults visiting OPD and IPD.

Organism isolated	Child					Adult				
	OPD/IPD				Total	OPD/IPD				Total
	OPD		IPD			OPD		IPD		
	No. of cases	%	No. of cases	%	No. of cases	No. of cases	%	No. of cases	%	No. of cases
MSSA	95	96.9%	4	100.0%	99	88	97.8%	19	95.0%	107
MRSA	3	3.1%	0	0.0%	3	2	2.2%	1	5.0%	3
Total	98	100.0%	4	100.0%	102	90	100.0%	20	100.0%	110
p-value	0.722					0.456				

MRSA was obtained in 3.1% pus samples of children and 2.2% pus samples in adults visiting OPD whereas in 5% pus samples of adults admitted in IPD.

MRSA obtained in either OPD or IPD were significantly different in both adults and children.

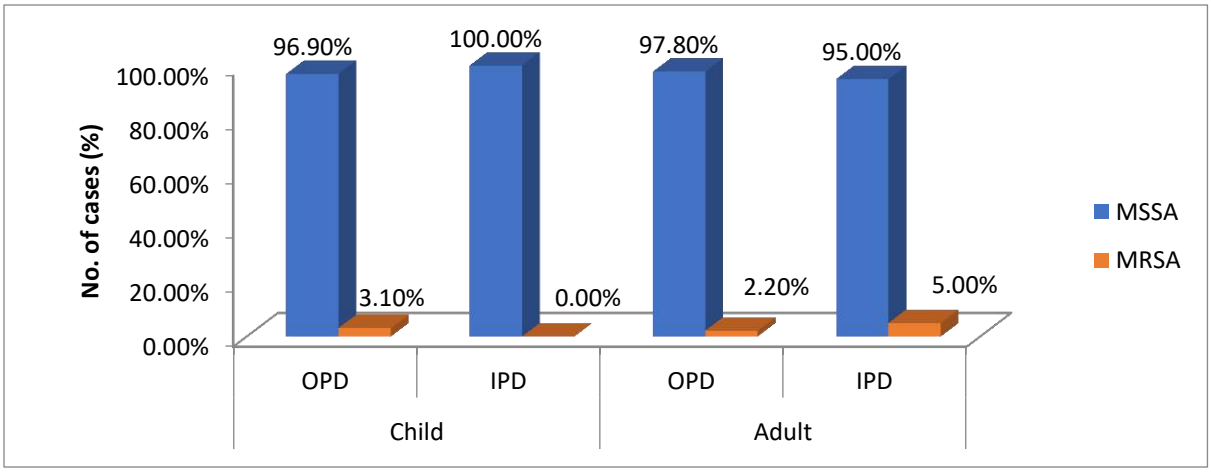


Figure 27: Comparison of MRSA isolated in child and adults visiting OPD and IPD.

Table 28: Comparison of correlation of bacteria isolated with primary pyoderma in children and adults.

organism isolated	Child		Adult		Total	
	No. of cases	%	No. of cases	%	No. of cases	%
S. aureus	62	90%	49	58%	111	72%
E. coli	2	3%	8	10%	10	6%
P. aeruginosa	1	1%	6	7%	7	4%
Enterococcus	1	1%	1	1%	2	2%
Enterobacter	0	0%	1	1%	1	1%
K. pneumoniae	0	0%	1	1%	1	1%
Proteus mirabilis	0	0%	1	1%	1	1%
Acinetobacter baumannii	0	0%	1	1%	1	1%
Citrobacter	0	0%	1	1%	1	1%
No growth	3	4%	15	18%	18	11%
Total	69	100%	84	100%	153	100%

In the primary pyodermas, the most common bacteria isolated was *S. aureus* in 62 (90%) children and 49 (58%) adults.

E. coli was seen in 2 (3%) primary pyoderma cases in children and 8 (10%) cases in adults.

P. aeruginosa was seen in 6 (7%) adults primary pyoderma cases and only 1 (1%) case in child.

No growth was obtained in 3 (4%) cases in children and 15 (18%) cases in adults.

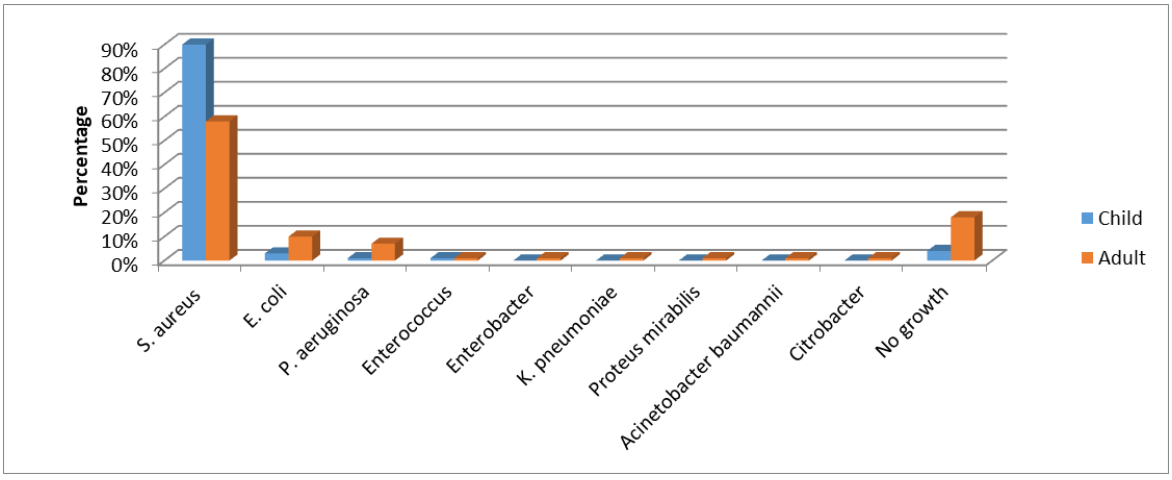


Figure 28: Comparison of correlation of bacteria isolated with primary pyoderma in children and adults.

Table 29: Comparison of correlation of bacteria isolated with secondary pyoderma in children and adults.

Organism isolated	Child		Adult		Total	
	No. of cases	%	No. of cases	%	No. of cases	%
S. aureus	26	79%	9	35%	35	59%
P. aeruginosa	0	0%	6	23%	6	10%
E. coli	2	6%	1	4%	3	5%
β-haemolytic streptococci	1	3%	1	4%	2	3%
K. pneumoniae	0	0%	1	4%	1	2%
Citrobacter	0	0%	1	4%	1	2%
Proteus vulgaris	0	0%	1	4%	1	2%
Enterobacter	0	0%	1	4%	1	2%
Enterococcus	1	3%	0	0%	1	2%
No growth	3	9%	5	19%	8	13%
Total	33	100%	26	100%	59	100%

The most common bacteria isolated in secondary pyodermas was *S. aureus* in 26 (79%) children and 9 (35%) adults.

P. aeruginosa was isolated only in 6 (23%) adults secondary pyoderma cases.

E. coli was obtained in 2 (6%) children and only in 1 (4%) (1) adult.

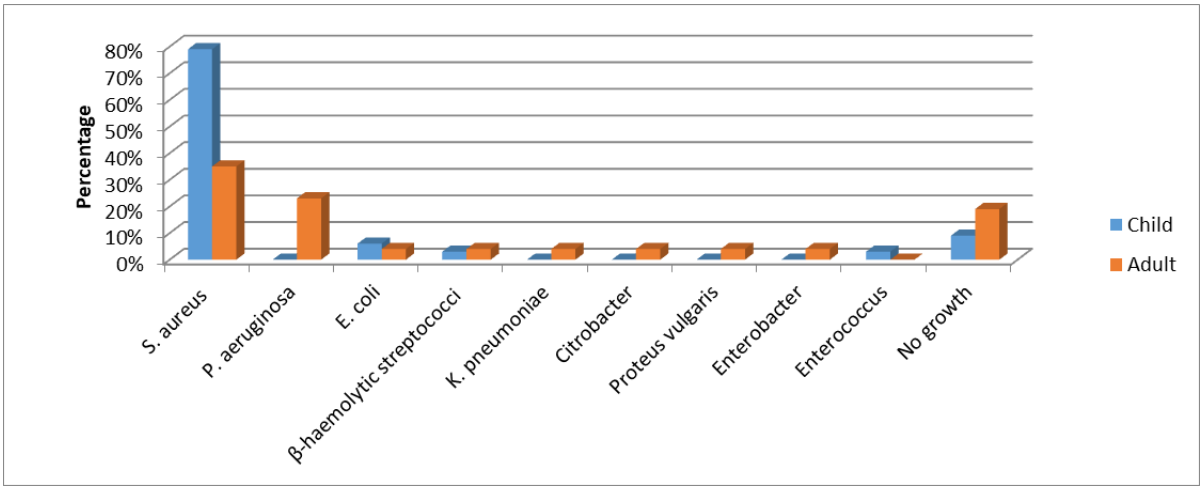


Figure 29: Comparison of correlation of bacteria isolated with secondary pyoderma in children and adults.

Table 30: Clinico-bacteriological analysis of gram positive organisms isolated with sub-types of primary pyoderma.

Clinical sub-types of Primary pyoderma	*S. aureus* (n=111)		*Enterococcus* (n=2)	
	No. of cases	%	No. of cases	%
Impetigo contagiosa	42	38%	0	0%
Folliculitis	25	23%	0	0%
Furuncle	15	14%	2	100%
Abscess	15	14%	0	0%
Carbuncle	4	4%	0	0%
Cellulitis	3	3%	0	0%
DCPA	2	2%	0	0%
Ecthyma	2	2%	0	0%
Sycosis barbae	2	2%	0	0%
Acute paronychia	1	1%	0	0%
Total	111	100%	2	100%

Maximum number of *S. aureus* was isolated in impetigo contagiosa (38%) followed by folliculitis (23%), furuncle (14%), abscess (14%), carbuncle (4%), cellulitis (3%) in decreasing order of frequency.

Enterococcus was seen only in furuncle (100%).

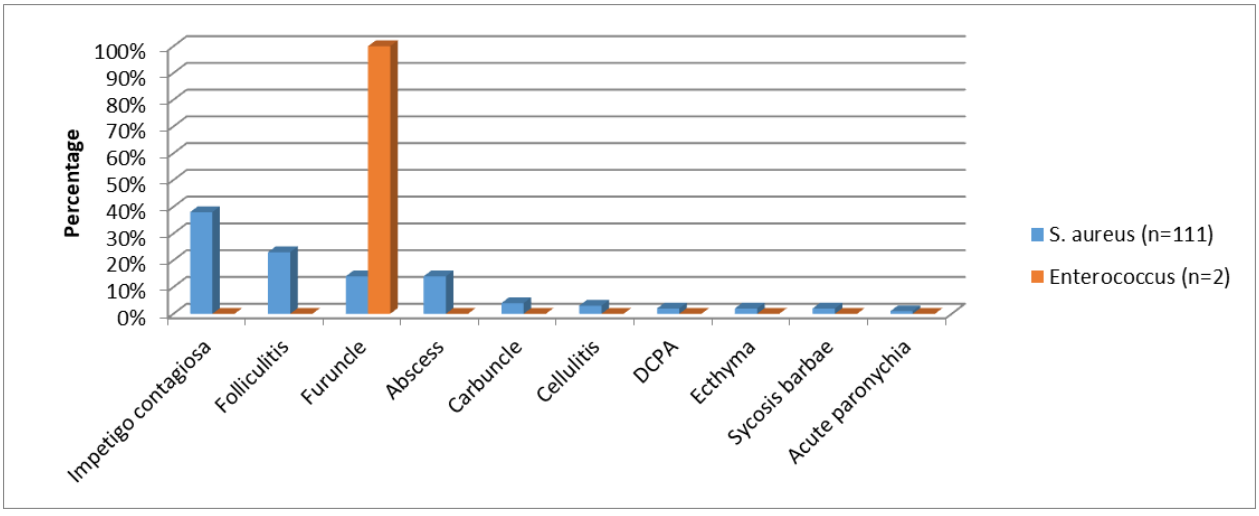


Figure 30: Clinico-bacteriological analysis of gram positive organisms isolated with sub-types of primary pyoderma.

Table 31: Clinico-bacteriological analysis of gram negative organisms isolated with sub-types of primary pyoderma.

Clinical sub-types of Primary pyoderma	*E. coli* (n=10)		*P. aeruginosa* (n=7)		Acinetobacter *baumannii* (n=1)		*Citrobacter* (n=1)		*Enterobacter* (n=1)		*K. pneumoniae* (n=1)		*Proteus mirabilis* (n=1)	
	no. of cases	%	no. of cases	%	no. of cases	%	no. of cases	%	no. of cases	%	no. of cases	%	No. of cases	%
Ecthyma	4	40%	1	14%	0	0%	0	0%	0	0%	0	0%	0	0%
Furuncle	2	20%	3	43%	0	0%	0	0%	0	0%	0	0%	1	100%
Carbuncle	2	20%	1	14%	0	0%	0	0%	0	0%	1	100%	0	0%
Abscess	1	10%	1	14%	1	100%	0	0%	0	0%	0	0%	0	0%
Cellulitis	1	10%	1	14%	0	0%	1	100%	1	100%	0	0%	0	0%
Total	10	100%	7	100%	1	100%	1	100%	1	100%	1	100%	1	100%

E. coli was seen in ecthyma (40%) followed by furuncle (20%), whereas *P. aeruginosa* in Furuncle (43%).

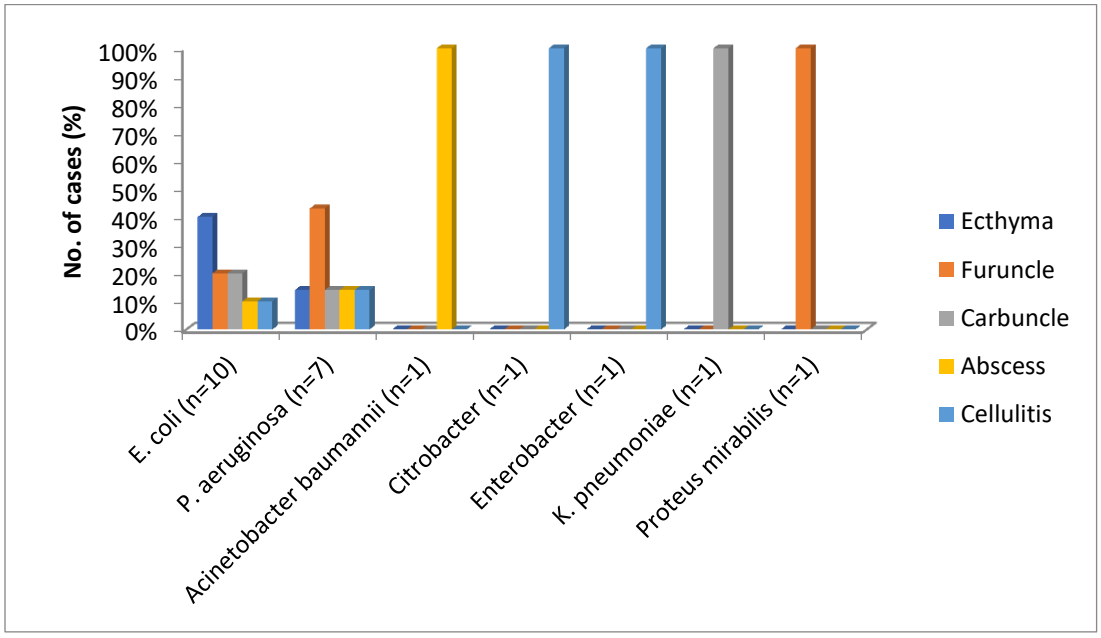


Figure 31: Clinico-bacteriological analysis of gram negative organisms isolated with sub-types of primary pyoderma.

Table 32: Clinico-bacteriological analysis of gram positive organisms isolated with sub-types of secondary pyoderma.

Clinical sub-types of Secondary pyoderma	*S. aureus* (n=35)		β -haemolytic streptococci (n=2)		*Enterococcus* (n=1)	
	No. of cases	%	No. of cases	%	No. of cases	%
Infected scabies	15	43%	1	50%	0	0%
Infected eczema	9	26%	0	0%	0	0%
Infected tinea	9	26%	1	50%	0	0%
Infected LSC	1	3%	0	0%	0	0%
Infected pediculosis	1	3%	0	0%	0	0%
Infected umbilical stump	0	0%	0	0%	1	100%
Total	35	100%	2	100%	1	100%

S. aureus was seen in infected scabies (43%) followed by infected eczema (26%).

β-haemolytic streptococci was seen only in Infected scabies (50%) and infected tinea (50%).

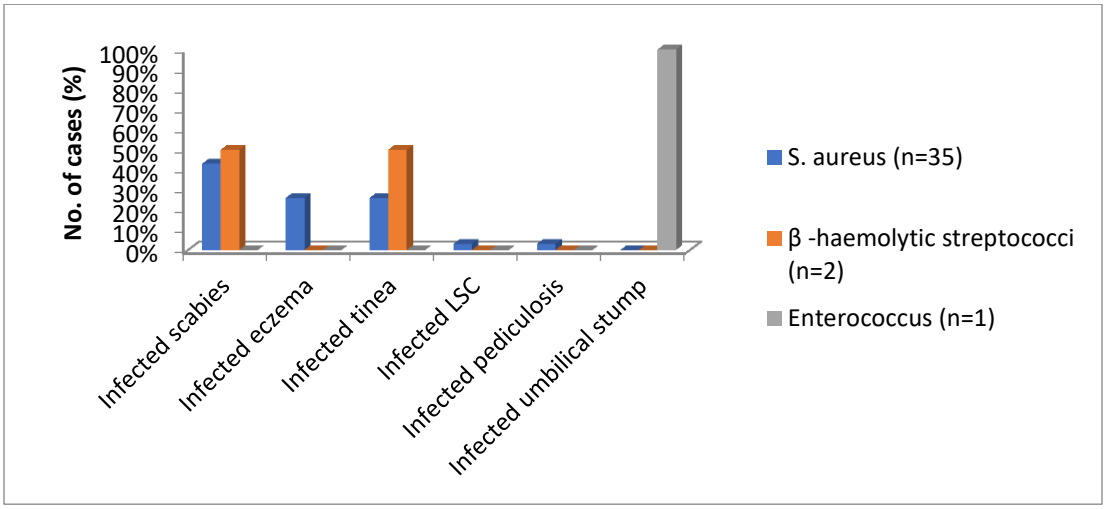


Figure 32: Clinico-bacteriological analysis of gram positive organisms isolated with sub-types of secondary pyoderma.

Table 33: Clinico-bacteriological analysis of gram negative organisms isolated with sub-types of secondary pyoderma.

Clinical sub-types of Secondary pyoderma	*E. coli* (n=3)		*P. aeruginosa* (n=6)		Enterobacter (n=1)		*Citrobacter* (n=1)		*K. pneumoniae* (n=1)		*Proteus vulgaris* (n=1)	
	No. of cases	%	No. of cases	%	No. of cases	%	No. of cases	%	No. of cases	%	No. of cases	%
Infected hemangioma	1	33%	0	0%	0	0%	0	0%	0	0%	0	0%
Infected LP	1	33%	0	0%	0	0%	0	0%	0	0%	0	0%
Infected umbilical stump	1	33%	0	0%	0	0%	0	0%	0	0%	0	0%
Infected tinea	0	0%	4	67%	0	0%	1	100 %	1	100%	0	0%
Infected eczema	0	0%	1	17%	0	0%	0	0%	0	0%	0	0%
Vasculitic ulcer	0	0%	1	17%	0	0%	0	0%	0	0%	1	100%
Infected LSC	0	0%	0	0%	1	100%	0	0%	0	0%	0	0%

E. coli (33%) was seen in Infected hemangioma, infected LP, Infected umbilical stump.

P. aeruginosa was seen in Infected tinea (67%).

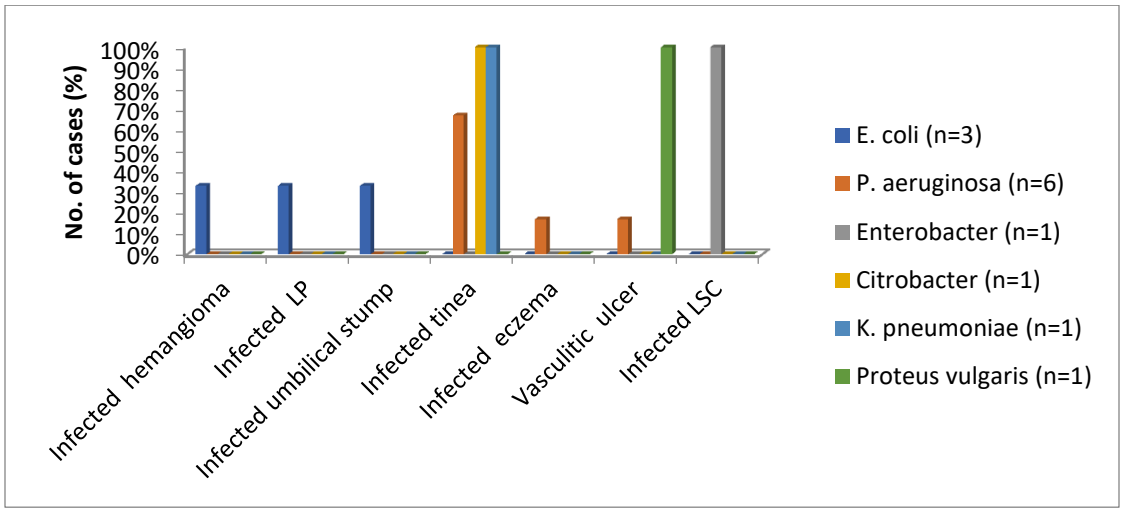


Figure 33: Clinico-bacteriological analysis of gram negative organisms isolated with sub-types of secondary pyoderma.

Table 34: Antibiotic susceptibility of gram positive bacteria.

Antibiotics	Sensitivity	*S. aureus* (n=146)		β -haemolytic streptococci (n=2)		*Enterococcus* (n=3)	
		No.	%	No.	%	No.	%
Cotrimoxazole	MS	4	3%	0	0%	0	0%
	R	14	10%	1	50%	0	0%
	S	124	85%	0	0%	0	0%
Penicillin	MS	2	1%	0	0%	0	0%
	R	71	49%	1	50%	0	0%
	S	72	49%	1	50%	3	100%
Amoxiclav	R	1	1%	0	0%	0	0%
	S	0	0%	0	0%	3	100%
Gentamycin	MS	3	2%	0	0%	0	0%
	R	10	7%	1	50%	0	0%
	S	131	90%	0	0%	3	100%
Clindamycin	R	8	5%	1	50%	0	0%
	S	137	95%	1	50%	0	0%
Erythromycin	MS	4	3%	0	0%	0	0%
	R	37	25%	2	100%	0	0%
	S	105	72%	0	0%	1	33%
Ciprofloxacin	MS	6	4%	0	0%	0	0%
	R	56	38%	1	50%	0	0%
	S	84	58%	0	0%	1	33%
Teicoplanin	R	3	2%	0	0%	0	0%
	S	143	98%	0	0%	3	100%
Vancomycin	R	0	0%	0	0%	0	0%
	S	146	100%	1	50%	3	100%
Linezolid	R	6	4%	0	0%	0	0%
	S	138	95%	2	100%	1	33%

Among all the isolates of *Staphylococcus*, maximum sensitivity i.e. 100% was seen to vancomycin followed by teicoplanin (98%) and linezolid (95%) and maximum resistance (49%) was seen to penicillin followed by ciprofloxacin (38%). Erythromycin which is an alternative drug to penicillin showed resistance in 25% of isolates.

Among B-haemolytic streptococci isolates, maximum sensitivity i.e. 100% was seen to linezolid and maximum resistance was seen to erythromycin (100%).

Among *Enterococcus* spp., maximum sensitivity i.e. 100% was seen to penicillin, amoxiclav, gentamycin, teicoplanin, vancomycin.

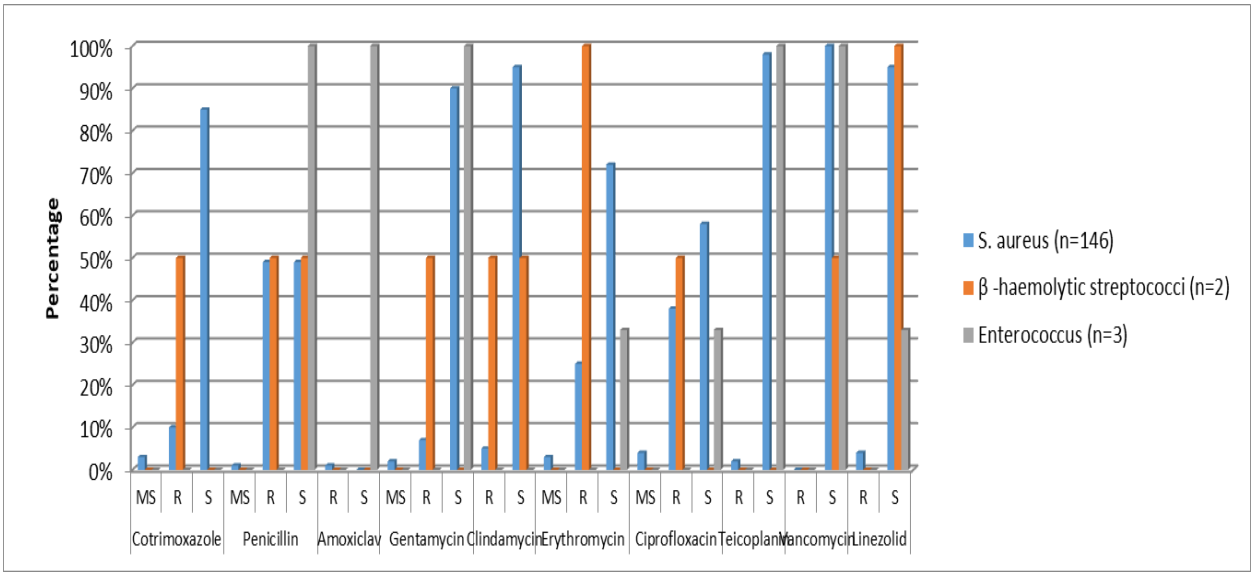


Figure 34: Antibiotic susceptibility of gram positive bacteria.

Table 35: Antibiotic susceptibility of gram negative bacteria.

Organism	Antibiotics	Ciprofloxacin			Gentamycin			Amikacin			Cefotaxime		Piperacillin			Piperacillin + Tazobactam		
sensitivity		MS	R	S	MS	R	S	MS	R	S	R	S	MS	R	S	MS	R	S
Acinetobacter baumannii (n=1)	No.	0	0	1	0	0	0	0	0	1	1	0	0	1	0	0	0	1
	%	0%	0%	100%	0%	0%	0%	0%	0%	100%	100%	0%	0%	100%	0%	0%	0%	100%
Citrobacter (n=2)	No.	0	0	2	0	0	1	0	0	2	0	1	0	0	1	0	0	2
	%	0%	0%	100%	0%	0%	50%	0%	0%	100%	0%	50%	0%	0%	50%	0%	0%	100%
E. coli(n=13)	No.	0	10	3	0	7	4	1	3	9	4	3	0	2	6	1	5	7
	%	0%	77%	23%	0%	54%	31%	8%	23%	69%	31%	23%	0%	15%	46%	8%	38%	54%
Enterobacter (n=2)	No.	0	0	2	0	0	2	0	0	2	0	1	0	0	0	0	0	2
	%	0%	0%	100%	0%	0%	100%	0%	0%	100%	0%	50%	0%	0%	0%	0%	0%	100%
K.pneumoniae (n=2)	No.	2	0	0	0	0	2	0	0	2	1	0	1	0	0	1	0	1
	%	100%	0%	0%	0%	0%	100%	0%	0%	100%	50%	0%	50%	0%	0%	50%	0%	50%
P. aeruginosa (n=13)	No.	0	3	9	0	2	11	0	2	11	0	0	0	0	10	0	2	11
	%	0%	23%	69%	0%	15%	85%	0%	15%	85%	0%	0%	0%	0%	77%	0%	15%	85%
Proteus mirabilis (n=1)	No.	0	1	0	0	0	1	0	0	1	0	0	0	0	0	0	0	1
	%	0%	100%	0%	0%	0%	100%	0%	0%	100%	0%	0%	0%	0%	0%	0%	0%	100%
Proteus vulgaris (n=1)	No.	0	0	1	0	0	1	0	0	1	0	1	0	0	0	0	0	1
	%	0%	0%	100%	0%	0%	100%	0%	0%	100%	0%	100%	0%	0%	0%	0%	0%	100%

100% sensitivity to piperacillin-tazobactam was observed in case of *Acinetobacter*, *Citrobacter, Enterobacte*r and *Proteus* except *Pseudomonas aeruginosa, E. coli* and *klebsiella* which showed 85%,54% and 50% respectively.

Acinetobacter spp. and *Citrobacter* spp. also showed 100% sensitivity to amikacin and ciprofloxacin.

Table 36: Antibiotic resistance in gram positive bacteria.

Gram positive bacteria	*S. aureus* (n=146)		β-haemolytic streptococci (n=2)	
Antibiotics	No.	%	No.	%
Cotrimoxazole	14	10%	1	50%
Penicillin	71	49%	1	50%
Amoxiclav	1	1%	0	0%
Gentamycin	10	7%	1	50%
Ciprofloxacin	56	38%	1	50%
Clindamycin	8	5%	1	50%
Erythromycin	37	25%	2	100%
Teicoplanin	3	2%	0	0%
Vancomycin	0	0%	0	0%
Linezolid	6	4%	0	0%

S. aureus showed resistance to penicillin (49%), ciprofloxacin (38%), erythromycin (25%), clindamycin (5%).

Resistance of β-haemolytic streptococci was seen to erythromycin (100%), cotrimoxazole (50%), penicillin (50%), gentamycin (50%), ciprofloxacin (50%), clindamycin (50%).

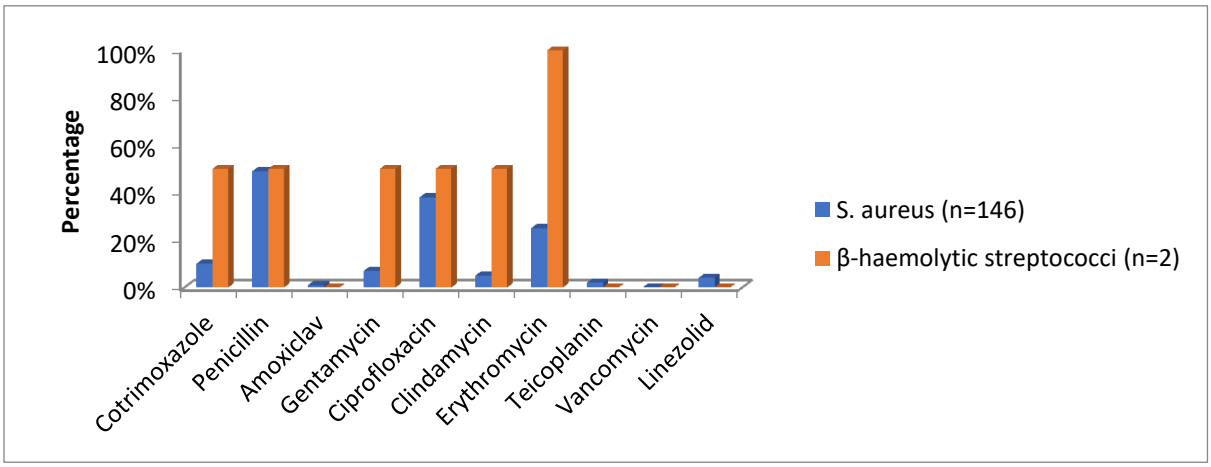


Figure 35: Antibiotic resistance in gram positive bacteria.

Table 37: Antibiotic resistance in gram negative bacteria.

Antibiotics	Amikacin	Ciprofloxacin	Gentamycin	Cefotaxime	Piperacillin	Piperacillin + Tazobactam
Gram negative bacteria	No.	%	No.	%	No.	%
E. coli (n=13)	23% (3)	77% (10)	54% (7)	31% (4)	15% (2)	38% (5)
P. aeruginosa (n=13)	15% (2)	23% (3)	15% (2)	0%	0%	15% (2)

Resistance of *E. coli* was seen to ciprofloxacin (77%), gentamycin (54%), piperacillin + tazobactam (38%), cefotaxime (31%), amikacin (23%).

P. aeruginosa showed resistance to ciprofloxacin (23%), gentamycin (15%), piperacillin+ tazobactam (15%) and amikacin (15%).

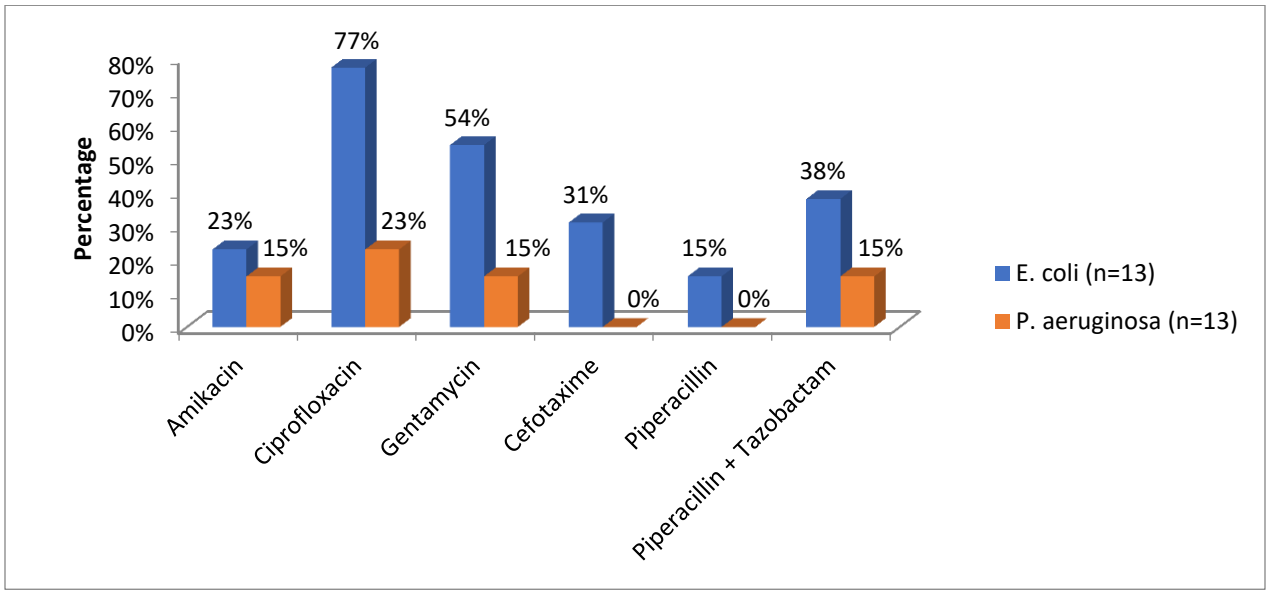


Figure 36: Antibiotic resistance in gram negative bacteria.

Table 38: Antibiotic resistance in gram positive bacteria in children.

Gram positive bacteria	*S. aureus* (n=88)		β -haemolytic streptococci (n=1)	
Antibiotics	No.	%	No.	%
Cotrimoxazole	3	3%	0	0%
Penicillin	33	38%	0	0%
Gentamycin	5	6%	0	0%
Ciprofloxacin	25	28%	0	0%
Clindamycin	5	6%	0	0%
Erythromycin	22	25%	1	100%
Teicoplanin	3	3%	0	0%
Vancomycin	0	0%	0	0%
Linezolid	0	0%	0	0%

Resistance of *S. aureus* was seen to penicillin (38%), ciprofloxacin (28%), erythromycin (25%).

β -haemolytic streptococci was completely resistant to erythromycin.

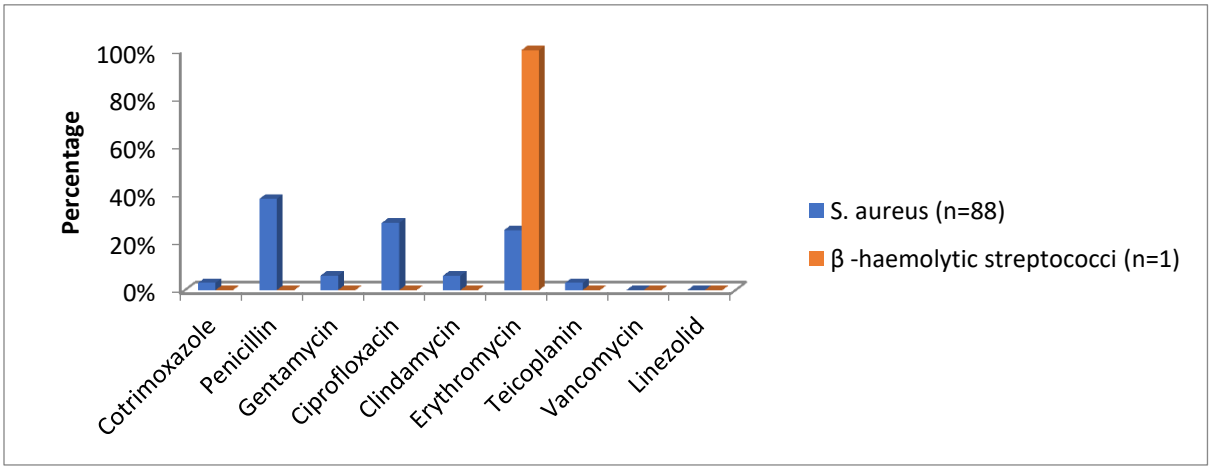


Figure 37: Antibiotic resistance in gram positive bacteria in children.

Table 39: Antibiotic resistance in gram positive bacteria in adult.

Gram positive bacteria	*S. aureus* (n=58)		β -haemolytic streptococci (n=1)	
Antibiotics	No.	%	No.	%
Cotrimoxazole	11	19%	1	100%
Penicillin	38	66%	1	100%
Gentamycin	5	9%	1	100%
Ciprofloxacin	31	53%	1	100%
Clindamycin	3	5%	1	100%
Erythromycin	15	26%	1	100%
Teicoplanin	0	0%	0	0%
Vancomycin	0	0%	0	0%
Linezolid	6	10%	0	0%

S. aureus showed resistance to penicillin (66%) followed by ciprofloxacin (53%), erythromycin (26%).

β-haemolytic streptococci showed complete resistance to cotrimoxazole, penicillin, gentamycin, clindamycin, ciprofloxacin and erythromycin.

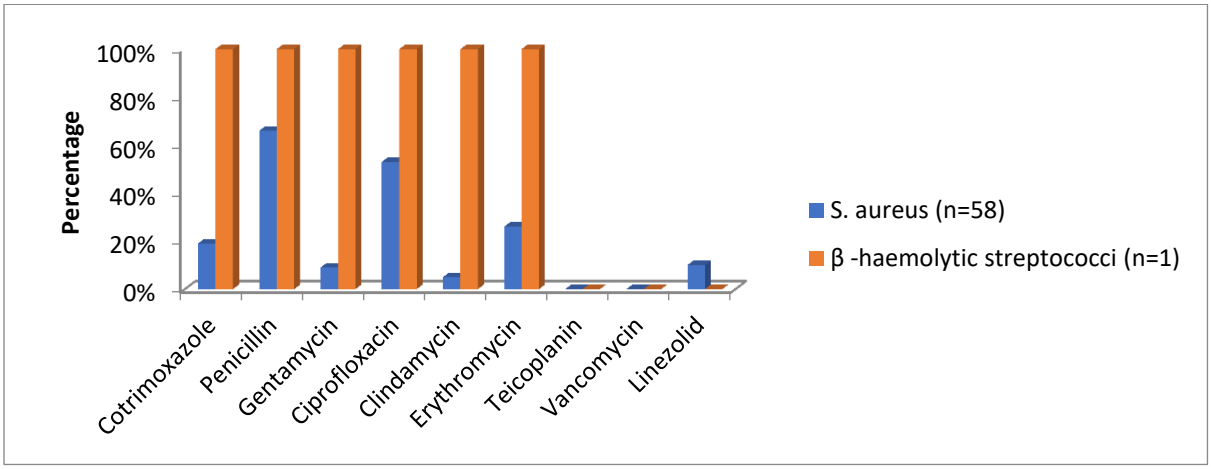


Figure 38: Antibiotic resistance in gram positive bacteria in adult.

Table 40: Antibiotic resistance in gram negative bacteria in children.

Gram negative bacteria	*E. coli* (n=4)		*P. aeruginosa* (n=1)	
Antibiotics	No.	%	No.	%
Amikacin	1	25%	1	100%
Ciprofloxacin	4	100%	1	100%
Gentamycin	3	75%	1	100%
Cefotaxime	2	50%	0	0%
Piperacillin	2	50%	0	0%
Piperacillin + Tazobactam	1	25%	1	100%
Cefoperazone + sulbactam	2	50%	1	100%
Ticarcillin + clavulanic acid	0	0	1	100%

Resistance of *E. coli* was seen to ciprofloxacin (100%) followed by gentamycin (75%).

Complete resistance of *P. aeruginosa* was seen to amikacin, ciprofloxacin, gentamycin, piperacillin + tazobactam, cefoperazone + sulbactam and ticarcillin + clavulanic acid.

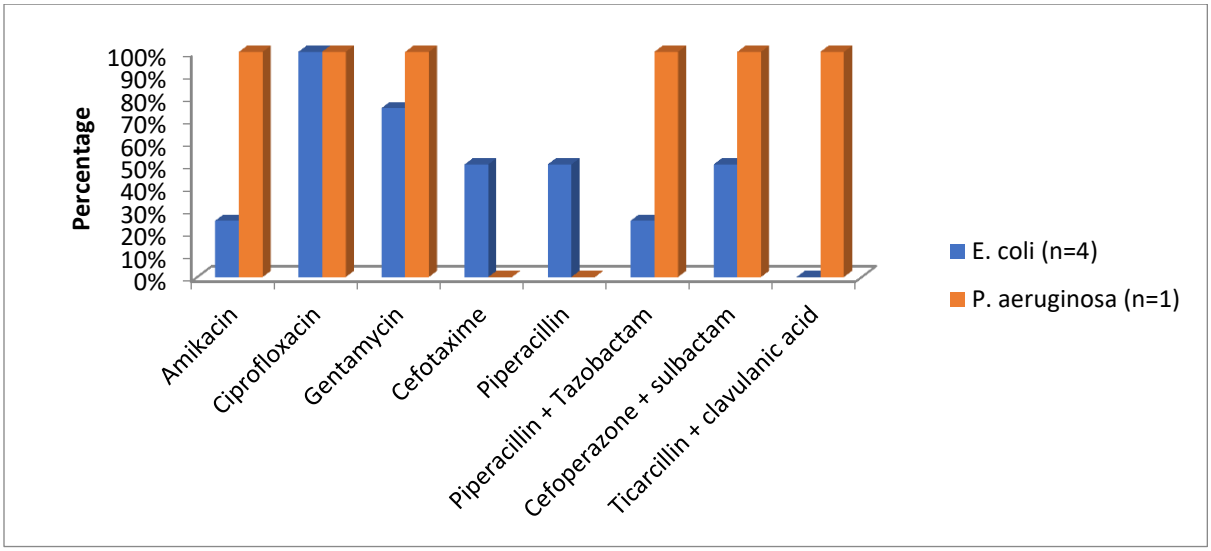


Figure 39: Antibiotic resistance in gram negative bacteria in children.

Table 41: Antibiotic resistance in gram negative bacteria in adult.

Gram negative bacteria	*E. coli* (n=9)		*P. aeruginosa* (n=12)	
Antibiotics	No.	%	No.	%
Amikacin	2	22%	1	8%
Ciprofloxacin	6	67%	2	17%
Gentamycin	4	44%	1	8%
Cefotaxime	2	22%	0	0%
Piperacillin	0	0%	0	0%
Piperacillin + Tazobactam	4	44%	1	8%
Cefoperazone + sulbactam	4	44%	2	17%
Ticarcillin + clavulanic acid	0	0%	1	8%

E. coli was seen to be resistant to ciprofloxacin (67%), gentamycin (44%), piperacillin + tazobactam (44%).

Resistance of *P. aeruginosa* was seen to ciprofloxacin and cefoperazone + sulbactam i.e. 17% each.

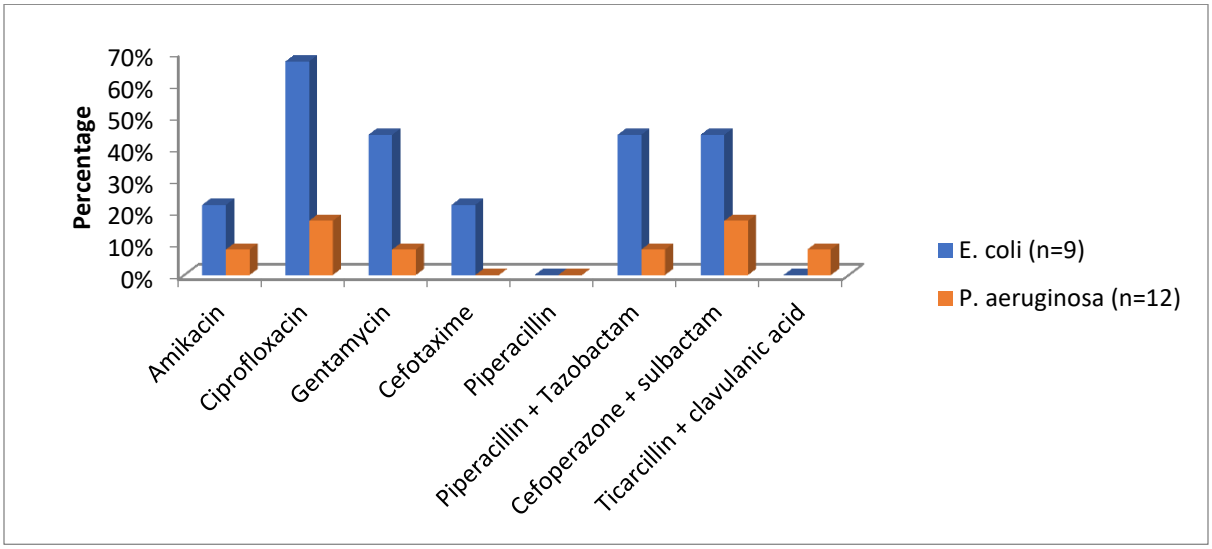


Figure 40: Antibiotic resistance in gram negative bacteria in adult.

Table 42: Antibiotic resistance in gram positive bacteria in OPD cases.

Gram positive bacteria	*S. aureus* (n=139)		β-haemolytic streptococci (n=2)	
Antibiotics	No.	%	No.	%
Cotrimoxazole	11	8%	1	50%
Penicillin	67	48%	1	50%
Gentamycin	9	6%	1	50%
Ciprofloxacin	51	37%	1	50%
Clindamycin	7	5%	1	50%
Erythromycin	33	24%	2	100%
Teicoplanin	3	2%	0	0%
Vancomycin	0	0%	0	0%
Linezolid	6	4%	0	0%

Resistance of *S. aureus* was seen to penicillin (48%) followed by ciprofloxacin (37%).

β-haemolytic streptococci was completely resistant to erythromycin.

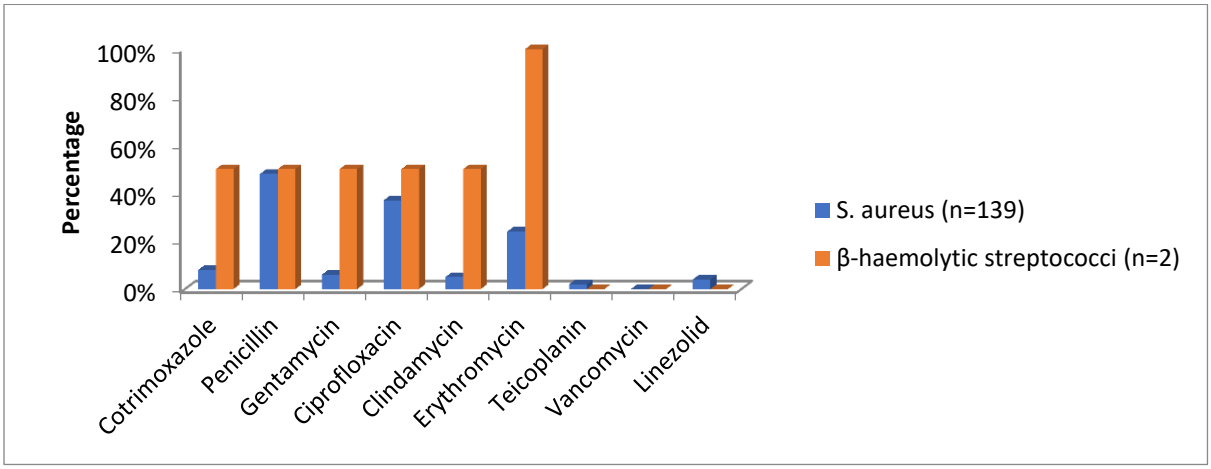


Figure 41: Antibiotic resistance in gram positive bacteria in OPD cases.

Table 43: Antibiotic resistance in gram positive bacteria in IPD cases.

Gram positive bacteria	*S. aureus* (n=7)	
Antibiotics	No.	%
Cotrimoxazole	3	43%
Penicillin	4	57%
Gentamycin	5	71%
Ciprofloxacin	5	71%
Clindamycin	1	14%
Erythromycin	4	57%
Teicoplanin	0	0%
Vancomycin	0	0%
Linezolid	0	0%

S. aureus showed resistance to gentamycin (71%), penicillin (57%), erythromycin (57%) in that order.

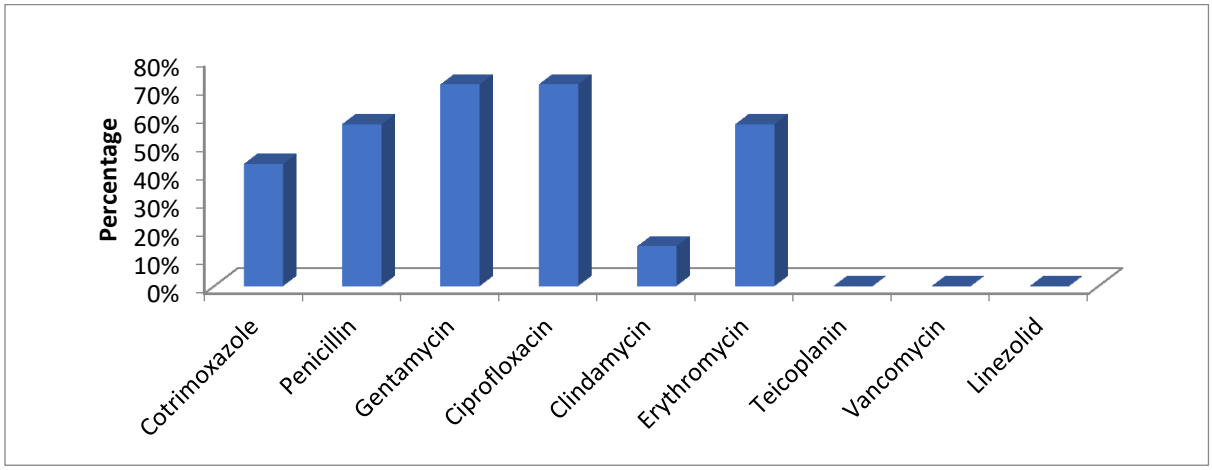


Figure 42: Antibiotic resistance in gram positive bacteria in IPD cases.

Table 44: Antibiotic resistance in gram negative bacteria in OPD cases.

Gram negative bacteria	*E. coli* (n=11)		*P. aeruginosa* (n=8)	
Antibiotics	No.	%	No.	%
Amikacin	2	18%	0	0%
Ciprofloxacin	8	73%	0	0%
Gentamycin	5	45%	0	0%
Cefotaxime	3	27%	0	0%
Piperacillin	2	18%	0	0%
Piperacillin + Tazobactam	4	36%	0	0%
Cefoperazone + sulbactam	5	45%	0	0%
Ticarcillin + clavulanic acid	0	0%	0	0%

Resistance of *E. coli* was seen to ciprofloxacin (73%) followed by gentamycin (45%).

P. aeruginosa was not resistant to any of the mentioned antibiotics among OPD patients.

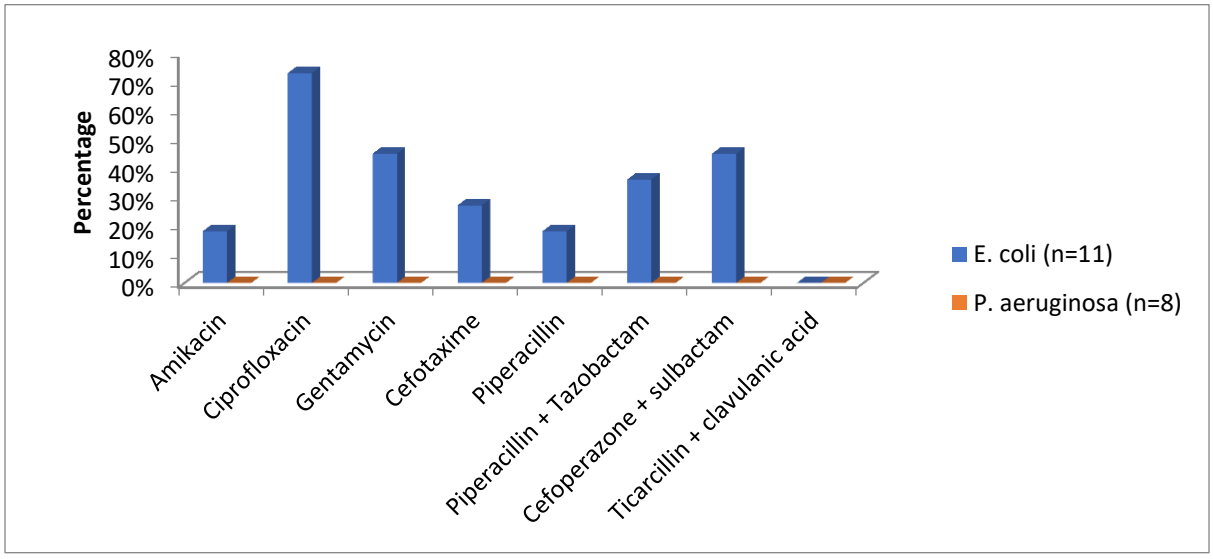


Figure 43: Antibiotic resistance in gram negative bacteria in OPD cases.

Table 45: Antibiotic resistance in gram negative bacteria in IPD cases.

Gram negative bacteria	*E. coli* (n=2)		*P. aeruginosa* (n=5)	
Antibiotics	No.	%	No.	%
Amikacin	1	50%	2	40%
Ciprofloxacin	2	100%	3	60%
Gentamycin	2	100%	2	40%
Cefotaxime	1	50%	0	0%
Piperacillin	0	0%	0	0%
Piperacillin + Tazobactam	1	50%	2	40%
Cefoperazone + sulbactam	1	50%	3	60%
Ticarcillin + clavulanic acid	0	0%	2	40%

Complete resistance of *E. coli* was seen to ciprofloxacin and gentamycin. *P. aeruginosa* was seen to be resistant to ciprofloxacin (60%) and cefoperazone + sulbactam (60%).

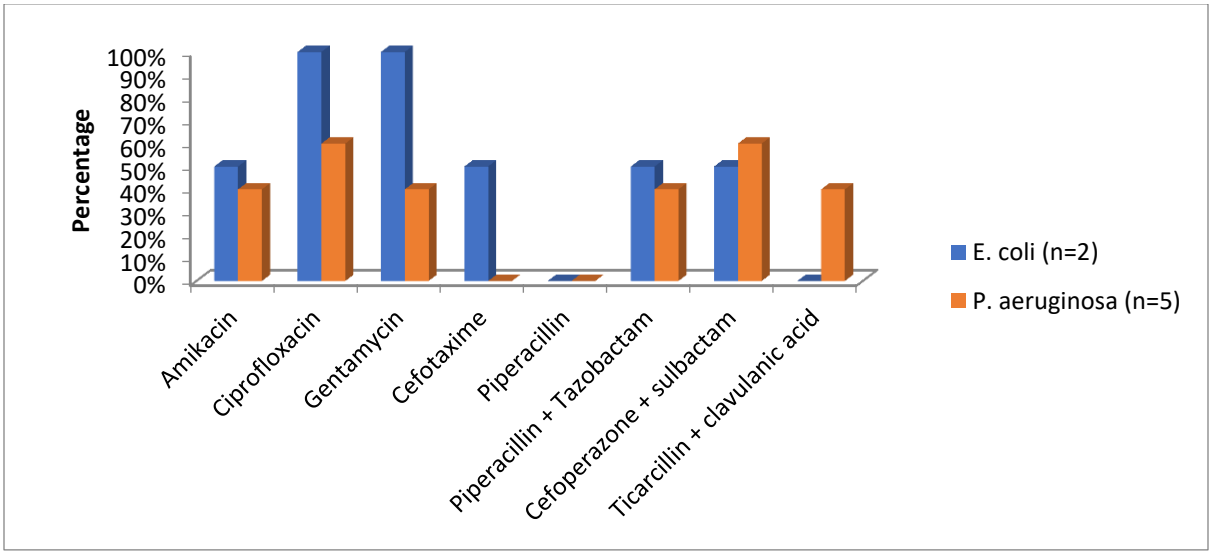


Figure 44: Antibiotic resistance in gram negative bacteria in IPD cases.

DISCUSSION

Pyodermas constitute a considerable proportion of cases worldwide and 17% of cases in India in dermatology clinics.[158] Multiple factors like overcrowding, illiteracy, poor socio-economic status and lack of personal hygiene are responsible for pyodermas.

The spectrum of organisms that causes pyodermas changes over time and varies from region to region. These organisms have also developed increasing multi drug resistance, thus the emergence of antibiotic resistant pathogenic bacteria is considered as grave threat to the public health worldwide.

During the last few decades, multidrug-resistant Gram-negative bacterial strains such as *Acinetobacter baumannii, E. coli, Klebsiella pneumoniae, Pseudomonas aeruginosa* and Gram-positive methicillin-resistant *Staphylococcus aureus* (MRSA) are associated with pus infections under hospital settings due to extensive overuse and inadequate dose regimen of antibiotics. It is a serious threat to public health globally due to the limited treatment options and discovery of new classes of antibiotics. Monitoring of resistance patterns in the hospital is needed to overcome these difficulties and to improve the outcome of serious infections in hospital settings.

The present study was undertaken to highlight the clinico-bacteriological correlation and antimicrobial susceptibility in children and adults with pyodermas. Therefore, the knowledge of the pattern of bacterial isolates and their antimicrobial susceptibility pattern is useful for prompt treatment of the patients.

Distribution of Patients

Two hundred patients were divided on the basis of age into two groups (children and adults), each comprising hundred patients. Out of hundred

children, 96 visited OPD and 4 were admitted in IPD. Similarly, out of hundred adults, 84 visited OPD and 16 were admitted in IPD.

Age Incidence

Pyodermas can affect any age group. In the present study of 200 clinically diagnosed cases of pyodermas, the age of patients ranged from 2 months to 86 years. There were 11% cases in adults of 2^{nd}, 4^{th} and 5^{th} decade of life each while majority of patients were seen in children of age group <10 years (34%) followed by 11-18 years (16%) which correlate well with study done by Gandhi et al.[159], which can be explained by the fact that preschool and school going children are prone to minor trauma caused mainly by insect bite.

Mean age of incidence in both groups was statistically different i.e. children (7.56 ± 5.24 years) and adults (46.13 ± 15.49 years). However, the study by Ashokan et al.[158] shows maximum cases in 1^{st} decade with mean age of incidence 34.67 years.

The least case incidence in our study was seen in 7^{th} decade (3%) comparable to the study by Harshita et al.[160]

Gender Incidence

In our study male preponderance was seen in both children and adults i.e. male (58%) and female (42%), with a M:F ratio of 1.4:1. In adults, males (56%) outnumbered females (44%). This is in concordance with studies done by Paudel et al.[161], Singh et al.[162], Ashokan et al.[158]

Similarly, in children pyodermas were more common in males (61%) than females (39%), which is comparable to the study done by Awal et al.[163] Male predominance in children is an incidental finding which may be due to random selection of cases and also due to regional variation. Another study by Hazarika et al.[164] have shown a female predominance in Pyoderma cases that is contradictory to our study.

Site of Involvement

Overall results of our study showed head and neck (34%) as the major site for pyoderma lesions, in contrast to the studies done by Ashokan et al.[158] and Paudel et al.[161] in which they observed lower limb was the commonly involved site in 37.90% and 29.30% patients respectively.

In both groups there were different sites involved i.e. head and neck (56%) in children and trunk (42%) in adults comparable to a study done by Hazarika et al.[164], that observed pyodermas over face in patients below 12 years of age. This is probably due to proximity of carrier sites like nares and throat are responsible for the increased incidence in head and neck area.

Clinical Manifestations of Pyodermas

The study showed that in adults pain was the predominant clinical feature (87%) followed by erythema (82%), swelling (54%) and oozing (30%) whereas in children crusting (70%) was prominent finding, which is in contrast to a study by Ashokan et al.[158], where oozing (85.30%) was more commonly seen, followed by pain (66.10%), and crusting (16.30%).

Associated Factors

There were many associated factors in our study including diabetes (16%), immunosuppression (9%) and poor hygiene (45%) which is comparable to a study by Singh et al.[162] which constitute 6.60% diabetic, 21.37% immunosuppressed and 23% poor hygienic patients. In adults, abscess (19%) was most common cause of pyoderma in diabetics in this study in contrast to the study by Singh et al.[162] furuncle (36.36%) was most commonly seen. In children, poor hygiene was associated with pyodermas (57%) in our study comparable (52%) with study by Hazarika et al.[164]. This proves that diabetes in adults and poor hygiene in children were significant associated features of pyodermas.

Type of Pyoderma

Primary pyodermas (72%) were more common than secondary pyodermas (28%). In primary pyodermas, 47.22% were children and 52.77% were adult and in secondary pyodermas, 57.14% were children and 42.85% were adults, in accordance to the studies done by Paudel et al.[161], Singh et al.[162], Mohan et al.[165] and Ashokan et al.[158]

In contrast to our study (20%), Malhotra et al.[166] showed a predominance of secondary (80.33%) pyoderma in his study of IPD patients.

Clinical Diagnosis

Host factors, such as integrity of the skin barrier with its acidic pH, presence of sebaceous secretions, lysozyme and production of defensins and adequate nutritional status plays an important role in prevention of infection. Trauma, insect bites, atopic dermatitis lead to disruption of skin barrier and increases the risk of primary pyodermas.

The overall pattern of primary pyoderma observed in our hospital shows impetigo as the single largest group which accounts for 30% (n=43) cases, followed by folliculitis with 24% (n=24), furunculosis (16%), abscess (13%), cellulitis and ecthyma (5%) each, acute paronychia (1%) (table no. 14.) This corresponds to the findings of Mohan et al.[165], Harshita et al.[160], whereas a study by Ashokan et al.[158] have ranked furunculosis (45.3%) as the commonest clinical diagnosis.

Primary pyoderma in children accounts for 63% cases of impetigo, contradictory to the findings of Awal et al.[163] (23.9%) and in adults folliculitis (29%) was most common, followed by abscess (22%) and furuncle (18%) in decreasing order of frequency, in contrast to the study by Singh et al.[162] who observed furuncle (27.2%) as the most common clinical diagnosis followed by folliculitis (13.2%).

The patients who visited OPD had folliculitis (26.7%) as the most common clinical diagnosis, comparable to the study by Paudel et al.[161] (26.7%)

whereas the patients recruited from IPD have furuncle (29%) as the most common finding contradictory to study of Malhotra et al.[166], who observed impetigo (14.75%) as commonest.

Certain dermatoses can be considered as a definite risk factor for secondary pyoderma. This is especially true for scabies, which is commonly superinfected, and, to a much lesser extent, tinea. Bacterial superinfection of scabies occurs commonly, and, according to the context and geographical area, varies from 16% to 67% of the cases, with similar bacteriological agents to those observed in primary pyoderma.

Secondary pyoderma in our study constituted 28% cases among two hundred patients. The common lesion observed in them were infected scabies 30% (n=17), infected tinea 30% (n=17), infected eczema 25%(n=14), vasculitis Ulcer 3%(n=2) (table no.16) whereas in the study by Harshita et al.[160], infected eczema (50.46%) was commonest.

Secondary pyoderma in children accounts for 47% infected scabies followed by 28% infected eczema, comparable to the study by Awal et al.[163] In adults, infected tinea (57%) was the most common clinical diagnosis which is contradictory to the study by Singh et al.[162] who observed IED (12.4%) as the commonest finding.

Among OPD patients, infected scabies (32%) was most commonly seen, contradictory to the study by Paudel et al.[161] who observed papular urticaria with secondary infection (14.7%) as commonest while in IPD patients, there was 1 case each of infected eczema (in children), infected tinea and vasculitic ulcer (in adults), in contrast to the study by Malhotra et al.[166], infected pemphigus (39.34%) was the commonest.

Type of Organism Obtained

Of the 200 patients subjected to gram staining and bacterial culture and sensitivity, Gram-positive organisms were cultured from 72% samples, whereas Gram-negative organisms were obtained from 12% samples.

There has been an increasing trend in the isolation of *S. aureus* as an etiological agent in pyoderma over the years. In our study, Gram-positive organisms (*S. aureus*- 77%) were the commonest isolate from pyoderma, also observed by Ashokan et al.[158] (55.70%) Mohan et al.[165] (83.90%) and Singh et al.[162] (77.83%).

Monomicrobial growth was seen in 82% pus samples, whereas polymicrobial growth was observed in 5% samples which is comparable to study by Malhotra et al.[166] Another study by Singh et al.[162] observed monomicrobial growth (94.34%) and polymicrobial growth in only 0.04% samples. No organism was seen in our study (13%), similar to the study by Malhotra et al.[166] (14.75%).

Isolated Organism

Identification of bacteria from skin lesion may provide important information as to the cause of cutaneous infections, whether primary or secondary to systemic processes. Only by correlating the clinical appearance of lesion with the bacterial data, one can reach the proper decision concerning the appearance of a bacterial disease.

Recent studies on Pyoderma indicate gram positive organism i.e. *Staphylococcus aureus* to be the main causative agent which is a significant change in the microbial trend of pyoderma. Previously *Streptococcus pyogenes* was the common etiological agent isolated.[166] Our study shows *Staphylococcus aureus* (77%) to be the major isolate in children and adults followed by *Enterococcus faecalis* 2%, *Streptococcus pyogenes* 1%. *S. aureus* was isolated in 72% primary pyodermas and 59% secondary pyodermas comparable to the studies by Singh et al.[162] and Ashokan et al.[158] MRSA was isolated in 3% children and 5% adults, which is in contrast to the studies by Harshita et al.[160] (42.20%) and Ashokan et al.[158] (47%).

Among gram negative bacteria *E. coli* (4%) in children and *P. aeruginosa* (14%) in adults were most commonly isolated which is comparable to the studies by Singh et al.[162] and Ashokan et al.[158] *E. coli* was seen more in primary (6%) as compared to secondary (5%) pyodermas which is in contrast to the studies by Singh et al.[162] and Ashokan et al.[158], they observed *E. coli* more in secondary than primary pyodermas.

Antibiotic Susceptibility

Over the past decades, there has been a rapid rise in antimicrobial resistance. To address this emerging antibiotic crisis, there is a need for developing treatment strategies that not only involves the development of new antibiotics, but also includes more optimal use of existing ones. Thus, a better understanding of bacteriological profile and its correlation to antibiotic susceptibility is essential. Antibacterial treatment in clinical infections is chosen on the basis of in vitro antibiotic susceptibility testing.

Antibiotic susceptibility testing was done over pus samples which revealed *S. aureus* was 100% sensitive to vancomycin followed by linezolid (96%) in our study, comparable to the study by Harshita et al.[160] It was resistant to penicillin in 49% cases in our study whereas in the study by Harshita et al.[160], it was resistant in 84.29%. *S. aureus* showed resistance to penicillin (66%) and ciprofloxacin (53%) in adults whereas in children resistance of *S. aureus* was seen to penicillin (38%) and ciprofloxacin (28%)

β-haemolytic streptococci showed complete resistance to cotrimoxazole, penicillin, gentamycin, clindamycin and ciprofloxacin in adults whereas in children it was 100% sensitive to these antibiotics.

E. coli was completely sensitive to piperacillin in adults whereas in children it was 50% sensitive. In our study, *E. coli* was resistant to ciprofloxacin (77%) followed by gentamycin (54%) which is in contrast to the study by Harshita et al.[160], in which it was resistant to ciprofloxacin

(37.50%) followed by gentamycin (25%), amikacin and piperacillin+ tazobactam in 6.25% each.

Complete resistance of *P. aeruginosa* was seen to amikacin, ciprofloxacin, gentamycin, piperacillin + tazobactam, cefoperazone + sulbactam and ticarcillin + clavulanic acid in children whereas in adults, resistance to ciprofloxacin and cefoperazone + sulbactam was seen in 17% each. In our study*, P. aeruginosa* showed resistance to ciprofloxacin (23%), amikacin (15%), piperacillin+ tazobactam (15%) and gentamycin (15%) comparable to the study by Harshita et al.[160] Harshita et al.[160] noticed *P. aeruginosa* showed resistance to ciprofloxacin (16.67%), gentamycin (16.67%), amikacin (8.34%), piperacillin+ tazobactam (8.34%).

SUMMARY AND CONCLUSIONS

A total of two hundred clinically diagnosed cases of pyodermas attending the outpatient and inpatient department of Dermatology of DMCH, Ludhiana were included in this study. They were divided into two groups on the basis of age. The first group consisted of 100 children of <18 years of age and second group consisted of 100 adults of >18 years of age.

- 90% patients visited OPD and 10% were admitted in IPD.
- It was observed that males outnumbered females with M:F ratio of 1.4:1. The most common age group affected was <10 years with 34% cases.
- The most common site of involvement was head and neck (56%) in children and trunk (42%) in adults.
- Adults presented with pain (87%) and erythema (82%) while in children, crusting (70%) was predominantly seen.
- Diabetes and poor hygiene were found to be significant risk factors associated with pyodermas.
- 32% adults affected with pyoderma were diabetic and presented mostly with abscess (19%).
- Poor hygiene was associated with pyodermas in 45% patients while 9% were immunosuppressed.
- 72% patients had primary pyodermas, among which impetigo contagiosa was the most common clinical presentation (63%) in children while folliculitis (29%) was seen in adults.
- Secondary pyodermas were seen in 28% patients, from which infected scabies (47%) ranked the highest in children while infected tinea (57%) in adults.
- Confirmation of the diagnosis by Gram's staining was done which revealed 72% pus samples had gram positive cocci while 12% had gram negative bacilli.
- Pus culture confirmation was done. *S. aureus* was the most common isolate in both children (92%) and adults (65%) among gram positive

organisms whereas among gram negative organisms, *E. coli* (4%) was seen in children whereas *P. aeruginosa* (14%) in adults.

- Gram positive organisms (88%) were mostly isolated from OPD patients whereas Gram negative organisms (67%) were mainly seen in IPD patients.
- *S. aureus* was isolated in 38% cases of impetigo.
- MRSA was isolated in 3% children and 5% adults.
- *E. coli* was seen in ecthyma (40%), whereas *P. aeruginosa* in furuncle (43%).
- Among all the isolates of *Staphylococcus*, maximum sensitivity i.e. 100% was seen to vancomycin followed by teicoplanin (98%) and linezolid (95%) and maximum resistance (49%) was seen to penicillin followed by ciprofloxacin (38%). Erythromycin which is an alternative drug to penicillin showed resistance in 25% of isolates.
- Resistance of *E. coli* was seen to ciprofloxacin (77%), gentamycin (54%), piperacillin + tazobactam (38%), cefotaxime (31%), amikacin (23%).
- *P. aeruginosa* showed resistance to ciprofloxacin (23%), gentamycin (15%), piperacillin+ tazobactam (15%) and amikacin (15%).

CONCLUSIONS

Pyodermas are frequently encountered in day to day clinical practice. Their management is complicated by the emergence of multidrug resistance amongst the commonly isolated etiological agents, thus limiting treatment options. Also, if not treated promptly, they are followed by various complications. Multidrug resistance has resulted from indiscriminate use of antibiotics. With knowledge of the likely causative organisms and their resistance patterns, proper antibiotic therapy can be given, thus avoiding unnecessary medication with drugs known to be resistant and also keeping newer antibiotics in reserve for use only against strains that are resistant to the common antibiotics.

Hence, timely recognition, and prompt bacterial diagnosis and antibiotic susceptibility testing is very important for the management of pyoderma and also to check for major complications.

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IMPETIGO

FOLLICULITIS

DERMATITIS CRURIS PUSTULOSA ET ATROPHICANS

SYCOSIS BARBAE

FURUNCLE

ABSCESS

ECTHYMA

CELLULITIS

PARONYCHIA

INFECTED TINEA

INFECTED PEDICULOSIS

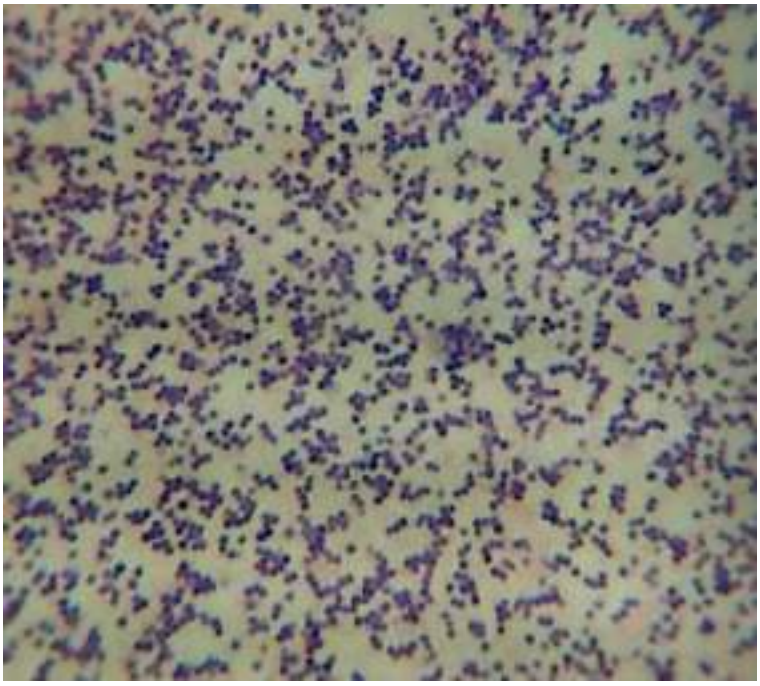
INFECTED SCABIES

INFECTED ECZEMA

INFECTED HEMANGIOMA

VASCULITIC ULCER

GRAM POSITIVE COCCI IN CLUSTERS



GROWTH OF *S. aureus* ON BLOOD AGAR

MAC CONKEY'S AGAR SHOWING GROWTH OF *E. coli*

VITEK 2 SYSTEM

PROFORMA

DATE-

Name :

S.NO.-

Age: Sex: MRD NO.-

Father/Husband's name:

Occupation:

Address:

Contact No:

Date of collection of sample:

Specimen:

Clinical presentation

-Primary/Secondary Pyoderma:

-Duration of disease:

Morphology of lesion- Site Size erythema
Crust(Color) Oozing(Color)

Symptoms- Redness Pain Swelling Other

Signature of Candidate **Signature of Supervisor**

Aggravating factors- Poor hygiene Diabetes

Immunosuppression Others

Nutrition and Built

History

-Past history

-Personal history

-Family history

-Treatment history

Clinical diagnosis

Microscopy

-Gram's staining

-Identification

Pus Culture-Isolated organism

Antibiotic susceptibility

Signature of Candidate **Signature of Supervisor**

KEY TO MASTER CHART

M	Male
F	Female
Y	Yes
N	No
P	Primary pyoderma
Sec	Secondary pyoderma
S	Sensitive
R	Resistant
MS	Moderately Sensitive
cm	Centimeter
OPD	Outpatient department
IPD	Inpatient department
LP	Lichen planus
EB	Epidermolysis bullosa
DCPA	Dermatitis cruris pustulosa et atrophicans
LSC	Lichen simplex chronicus
S. aureus	*Staphylococcus aureus*
P. aeruginosa	*Pseudomonas aeruginosa*
E. coli	*Escherichia coli*
K. pneumoniae	*Klebsiella pneumoniae*
MRSA	Methicillin-resistant *Staphylococcus aureus*

S.No.	MRD No.	Name	Age (years)	Sex	OPD/ IPD	Clinical presentation			Risk factors			Symptoms			Morphology of lesions			
						Duration of Disease (days)	Site	Size (cm)	Poor hygiene	H/o Diabetes	Immunosuppression	Redness	Pain	Swelling	Erythema	Crusting	Crusting (color)	Oozing
1	577690	P	11	F	OPD	10	Head and neck	1	Y	N	N	N	Y	Y	N	Y	Yellowish-brown	N
2	646841	R	11	M	OPD	5	Upper limb	1	N	N	N	Y	Y	N	Y	Y	Yellowish-brown	Y
3	642858	R	0.4	M	OPD	5	Lower limb	2	N	N	N	Y	Y	N	Y	N	-	Y
4	761055	MS	0.5	M	OPD	3	Lower limb	0.5	N	N	N	Y	Y	N	Y	N	-	N
5	690890	H	18	M	OPD	6	Trunk	0.3	N	N	N	Y	Y	N	Y	N	-	N
6	701900	VS	14	M	OPD	7	Head and neck	2	Y	N	Y	Y	Y	Y	Y	N	-	N
7	762029	MK	16	F	OPD	7	Lower limb	2	N	N	N	N	Y	Y	N	N	-	Y
8	580416	H	8	M	OPD	3	Upper limb	0.2	Y	N	N	Y	Y	Y	Y	N	-	N
9	366446	H	17	M	OPD	4	Upper limb	0.3	N	N	Y	Y	Y	N	Y	N	-	Y
10	721514	G	0.2	F	OPD	1	Trunk	0.3	Y	N	N	Y	Y	N	Y	Y	Honey colored	N
11	736449	V	18	M	OPD	6	Head and neck	1	Y	N	Y	Y	Y	Y	Y	N	-	Y
12	578907	KS	9	M	IPD	6	Head and neck	0.3	Y	N	N	Y	Y	N	Y	Y	Honey colored	Y
13	739951	RG	13	M	OPD	7	Upper limb	0.2	N	N	N	Y	Y	N	Y	Y	Honey colored	N
14	543859	TS	11	F	IPD	2	Upper limb	0.3	N	N	Y	Y	Y	Y	Y	N	-	N
15	589064	J	4	F	OPD	7	Upper limb	1	N	N	N	Y	Y	N	Y	Y	Honey colored	Y
16	602955	AS	18	F	OPD	3	Head and neck	1	Y	N	N	Y	Y	Y	Y	Y	Dirty brown	Y
17	82730	S	3	M	OPD	2	Head and neck	0.3	Y	N	N	Y	Y	N	Y	Y	Honey colored	N
18	712413	CN	1	M	OPD	1	Head and neck	0.5	N	N	N	Y	Y	N	Y	Y	Honey colored	N
19	806888	S	1	M	OPD	7	Upper limb	0.5	Y	N	N	N	Y	N	N	Y	Dirty brown	Y
20	647694	R	1	M	OPD	3	Head and neck	0.5	Y	N	N	N	N	N	N	Y	Dirty brown	Y
21	531797	JS	2	M	OPD	4	Head and neck	0.4	Y	N	N	N	N	N	N	Y	Dirty brown	Y
22	823357	T	8	F	OPD	7	Head and neck	0.5	Y	N	Y	N	N	N	N	Y	Dirty brown	Y
23	823837	SS	4	M	OPD	2	Trunk	0.4	Y	N	N	Y	Y	N	Y	Y	Honey colored	N
24	810330	AS	12	F	OPD	8	Head and neck	0.5	Y	N	N	N	N	N	N	Y	Reddish-yellow	N
25	827246	G	6	M	OPD	2	Head and neck	0.5	N	N	N	Y	Y	N	Y	Y	Honey colored	N
26	822305	AV	9	M	OPD	7	Head and neck	0.4	Y	N	Y	Y	Y	N	Y	Y	Dirty brown	Y
27	830656	IK	2	M	OPD	7	Head and neck	0.5	N	N	N	Y	Y	N	Y	Y	Honey colored	N
28	596031	DD	3	M	OPD	2	Upper limb	0.5	N	N	N	Y	Y	N	Y	Y	Honey colored	N
29	720234	SK	7	M	OPD	4	Head and neck	0.5	N	N	N	Y	Y	N	Y	Y	Honey colored	N
30	867723	CK	14	M	OPD	3	Trunk	0.3	N	N	N	Y	Y	N	Y	N	-	N
31	840074	DJ	5	M	OPD	3	Upper limb	0.5	N	N	N	N	Y	N	N	Y	Yellowish-brown	N

S.No.	MRD No.	Oozing (color)	Type of pyoderma	Clinical Diagnosis	Gram's staining	Identification		MRSA	Cotrimoxazole	Amikacin	Cefoxitin	Ciprofloxacin
						organism	organism					
1	577690	-	Sec	Infected pediculosis	many pus cells	S.aureus	-	-	S	S	S	R
2	646841	Yellow	P	Furuncle	gram positive cocci,few pus cells	S.aureus	Enterococcus	-	S	S	S	S
3	642858	Yellow	Sec	Infected hemangioma	gram negative bacilli,many pus cells	E.coli	-	-	R	S	-	R
4	761055	-	Sec	Infected umblical stump	gram positive cocci,gram negative bacilli,few pus cells	E.coli	Enterococcus	-	R	S	-	R
5	690890	-	P	Folliculitis	few pus cells	no growth	-	-	-	-	-	-
6	701900	-	P	Carbuncle	gram positive cocci in cluster,many pus cells	S.aureus	-	-	S	S	S	S
7	762029	Yellow	P	Abscess	gram positive cocci in cluster,many pus cells	S.aureus	-	-	-	S	-	R
8	580416	-	P	Folliculitis	gram positive cocci in cluster,many pus cells	S.aureus	-	Y	S	S	R	R
9	366446	Yellow	Sec	Infected EB	many pus cells	no growth	-	-	-	-	-	-
10	721514	-	P	Impetigo contagiosa	gram positive cocci in cluster,many pus cells	S.aureus	-	-	S	S	S	S
11	736449	Yellow	Sec	Infected scabies	gram positive cocci in cluster,many pus cells	S.aureus	-	-	MS	S	S	S
12	578907	Yellow	P	Impetigo contagiosa	many pus cells	no growth	-	-	-	-	-	-
13	739951	-	P	Impetigo contagiosa	gram positive cocci in cluster,many pus cells	S.aureus	-	-	S	S	S	R
14	543859	-	P	Furuncle	gram negative bacilli,many pus cells	P.aeruginosa	-	-	-	R	-	R
15	589064	Yellow	P	Impetigo contagiosa	gram positive cocci in cluster,many pus cells	S.aureus	-	-	S	S	S	R
16	602955	Yellow	Sec	Infected tinea	gram positive cocci in cluster,many pus cells	S.aureus	-	-	S	S	S	R
17	82730	-	P	Impetigo contagiosa	gram positive cocci in cluster,many pus cells	S.aureus	-	-	R	S	S	S
18	712413	-	P	Impetigo contagiosa	gram positive cocci in cluster,many pus cells	S.aureus	-	-	S	S	S	S
19	806888	Yellow	Sec	Infected scabies	Gram positive cocci in chains,few pus cells	S.hemolyticus	-	-	-	-	-	-
20	647694	Yellow	Sec	Infected scabies	gram positive cocci in cluster,many pus cells	S.aureus	-	-	-	S	-	R
21	531797	Yellow	Sec	Infected scabies	gram positive cocci in cluster,many pus cells	S.aureus	-	-	S	S	S	S
22	823357	Yellow	Sec	Infected scabies	gram positive cocci in cluster,many pus cells	S.aureus	-	-	S	S	S	S
23	823837	-	P	Impetigo contagiosa	gram positive cocci in cluster,few pus cells	S.aureus	-	-	S	S	S	S
24	810330	-	P	Folliculitis	gram positive cocci in cluster,many pus cells	S.aureus	-	-	S	S	S	R
25	827246	-	P	Impetigo contagiosa	gram positive cocci in cluster,many pus cells	S.aureus	-	-	S	S	S	S
26	822305	Yellow	Sec	Infected scabies	gram positive cocci in cluster,many pus cells	S.aureus	-	-	S	S	S	S
27	830656	-	P	Impetigo contagiosa	gram positive cocci in cluster,few pus cells	S.aureus	-	-	S	S	S	S
28	596031	-	P	Impetigo contagiosa	gram positive cocci in cluster,many pus cells	S.aureus	-	-	S	S	S	S
29	720234	-	P	Impetigo contagiosa	gram positive cocci in cluster,few pus cells	S.aureus	-	-	-	S	-	R
30	867723	-	Sec	Infected tinea	gram positive cocci in cluster,many pus cells	S.aureus	-	-	S	S	S	S
31	840074	-	Sec	Infected eczema	gram positive cocci in cluster,many pus cells	S.aureus	-	-	S	S	S	S

S.No.	MRD No.	Antibiotic susceptibility													
		Clindamycin	Erythromycin	Gentamycin	Linezolid	Penicillin	Teicoplanin	Vancomycin	Cefotaxime	Amoxiclav	Cefoperazone + Sulbactam	Piperacillin	Piperacillin+Tazobactam	Oxacillin	Ticarcillin+clavulanic acid
1	577690	S	S	S	S	R	S	S	-	-	-	-	-	-	-
2	646841	S	S	S	S	S	S	S	-	S	-	-	-	-	-
3	642858	-	-	R	-	-	-	-	R	-	R	R	S	-	-
4	761055	-	-	R	-	-	-	-	R	-	R	R	S	-	-
5	690890	-	-	-	-	-	-	-	-	-	-	-	-	-	-
6	701900	S	S	S	S	S	S	S	-	-	-	-	-	-	-
7	762029	R	R	S	S	R	S	S	-	-	-	-	-	-	-
8	580416	S	R	S	S	R	R	S	-	-	-	-	-	-	-
9	366446	-	-	-	-	-	-	-	-	-	-	-	-	-	-
10	721514	S	S	S	S	S	S	S	-	-	-	-	-	-	-
11	736449	S	S	MS	S	R	S	S	-	-	-	-	-	-	-
12	578907	-	-	-	-	-	-	-	-	-	-	-	-	-	-
13	739951	S	S	S	S	R	S	S	-	-	-	-	-	-	-
14	543859	-	-	R	-	-	-	-	-	-	R	-	R	-	R
15	589064	S	R	R	S	R	S	S	-	-	-	-	-	-	-
16	602955	S	S	S	S	S	S	S	-	-	-	-	-	-	-
17	82730	S	S	S	S	R	S	S	-	-	-	-	-	-	-
18	712413	S	S	S	S	S	S	S	-	-	-	-	-	-	-
19	806888	S	R	-	S	S	-	S	S	-	-	-	-	-	-
20	647694	R	R	S	S	R	S	S	-	-	-	-	-	-	-
21	531797	S	S	S	S	R	S	S	-	-	-	-	-	-	-
22	823357	S	R	S	S	R	S	S	-	-	-	-	-	-	-
23	823837	S	S	S	S	S	S	S	-	-	-	-	-	-	-
24	810330	S	S	S	S	S	S	S	-	-	-	-	-	-	-
25	827246	S	S	S	S	S	S	S	-	-	-	-	-	-	-
26	822305	S	R	S	S	R	S	S	-	-	-	-	-	-	-
27	830656	S	S	S	S	S	S	S	-	-	-	-	-	-	-
28	596031	S	R	S	S	R	S	S	-	-	-	-	-	-	-
29	720234	R	R	S	S	R	S	S	-	-	-	-	-	-	-
30	867723	S	MS	S	S	R	S	S	-	-	-	-	-	-	-
31	840074	S	S	S	S	S	S	S	-	-	-	-	-	-	-

						Clinical presentation			Risk factors			Symptoms			Morphology of lesions			
S.No.	MRD No.	Name	Age (years)	Sex	OPD/ IPD	Duration of Disease (days)	Site	Size (cm)	Poor hygiene	H/o Diabetes	Immunosuppression	Redness	Pain	Swelling	Erythema	Crusting	Crusting (color)	Oozing
32	839745	MK	10	F	OPD	9	Head and neck	0.4	N	N	N	N	Y	N	N	N	-	N
33	754276	KV	1	F	OPD	6	Trunk	0.5	N	N	N	N	Y	N	N	N	-	N
34	71801	A	18	M	OPD	7	Trunk	1	Y	N	N	Y	Y	Y	Y	N	-	N
35	844596	J	2	M	OPD	4	Head and neck	0.5	Y	N	N	Y	N	N	Y	Y	Dirty brown	Y
36	864744	A	16	M	OPD	3	Upper limb	0.5	N	N	N	Y	Y	N	Y	N	-	N
37	861854	S	9	F	OPD	7	Trunk	0.5	Y	N	N	Y	Y	N	Y	Y	Yellowish-brown	N
38	624123	NK	12	F	OPD	1	Head and neck	0.5	N	N	Y	Y	Y	N	Y	N	-	Y
39	858546	PK	11	M	OPD	2	Upper limb	0.5	N	N	N	Y	Y	N	Y	Y	Honey colored	N
40	892443	SA	16	F	OPD	6	Trunk	0.5	Y	N	N	N	Y	N	N	Y	Dirty brown	N
41	859772	HS	6	M	OPD	4	Head and neck	0.5	Y	N	N	N	Y	N	N	Y	Dirty brown	N
42	245182	MS	16	M	OPD	7	Lower limb	1	Y	N	Y	Y	Y	Y	Y	Y	Dirty brown	N
43	57891	JS	5	M	OPD	3	Head and neck	0.4	N	N	N	Y	Y	N	Y	Y	Honey colored	N
44	866147	IK	12	F	IPD	6	Lower limb	0.4	N	N	N	Y	N	N	Y	Y	Dirty brown	N
45	532997	SM	3	M	OPD	9	Head and neck	0.5	N	N	N	Y	N	N	Y	Y	Dirty brown	N
46	866792	S	1	M	OPD	4	Trunk	0.5	Y	N	N	Y	Y	N	Y	Y	Yellowish-brown	N
47	875738	A	14	M	OPD	8	Trunk	0.4	Y	N	N	Y	N	N	Y	N	-	N
48	848220	PS	16	M	OPD	5	Upper limb	1	N	N	N	Y	Y	N	Y	Y	Dirty brown	N
49	88971	PS	15	M	OPD	4	Lower limb	2	N	N	N	Y	N	N	Y	N	-	N
50	428622	M	13	F	OPD	1	Head and neck	0.5	N	N	N	Y	Y	N	Y	Y	Honey colored	N
51	911534	N	3	F	OPD	7	Head and neck	0.5	Y	N	N	N	N	N	N	N	-	N
52	881931	YK	3	F	OPD	6	Trunk	0.5	Y	N	N	Y	Y	N	Y	N	-	N
53	901862	NS	5	M	OPD	5	Trunk	0.4	N	N	N	Y	N	N	Y	N	-	N
54	910036	U	15	M	OPD	7	Upper limb	0.5	N	N	N	Y	N	N	Y	N	-	N
55	910035	AS	15	M	OPD	2	Head and neck	0.3	N	N	N	Y	Y	N	Y	Y	Honey colored	N
56	643229	SS	13	M	OPD	10	Head and neck	0.4	N	N	N	y	y	y	y	Y	Dirty brown	Y
57	784587	J	2	F	OPD	1	Head and neck	0.4	N	N	N	Y	Y	N	Y	Y	Honey colored	N
58	866738	AA	5	M	OPD	4	Head and neck	0.4	Y	N	N	N	Y	N	N	Y	Dirty brown	N
59	859772	HS	6	M	OPD	7	Head and neck	0.5	Y	N	N	N	Y	N	N	Y	Dirty brown	N
60	201778	J	5	F	OPD	6	Trunk	0.5	Y	N	N	N	N	N	N	N	-	N
61	891931	MS	15	M	IPD	10	Head and neck	0.5	N	N	N	Y	Y	N	Y	N	-	N
62	893020	SS	4	M	OPD	2	Head and neck	0.3	N	N	N	Y	Y	N	Y	Y	Honey colored	N
63	892443	SA	15	F	OPD	3	Upper limb	0.5	N	N	N	Y	Y	N	Y	Y	Honey colored	N
64	384767	RG	7	M	OPD	5	Head and neck	0.5	Y	N	N	N	Y	N	N	Y	Dirty brown	N
65	905126	VK	4	F	OPD	5	Head and neck	0.4	Y	N	N	N	Y	N	N	Y	Dirty brown	N
66	883401	PJ	9	F	OPD	4	Upper limb	0.5	Y	N	N	N	N	N	N	N	-	N
67	211879	VG	4	M	OPD	1	Head and neck	0.3	N	N	N	Y	Y	N	Y	Y	Honey colored	N

S.No.	MRD No.	Oozing (color)	Type of pyoderma	Clinical Diagnosis	Gram's staining	Identification		MRSA				
						organism	organism		Cotrimoxazole	Amikacin	Cefoxitin	Ciprofloxacin
32	839745	-	P	Folliculitis	gram positive cocci in cluster,many pus cells	S.aureus	-	-	S	S	S	S
33	754276	-	P	Folliculitis	gram positive cocci in cluster,few pus cells	S.aureus	-	-	S	S	S	S
34	71801	-	P	Cellulitis	gram positive cocci in cluster,many pus cells	S.aureus	-	-	S	S	S	S
35	844596	Yellow	Sec	Infected scabies	gram positive cocci in cluster,many pus cells	S.aureus	-	-	S	S	S	S
36	864744	-	P	Folliculitis	gram positive cocci in cluster,many pus cells	S.aureus	-	-	S	S	S	S
37	861854	-	Sec	Infected scabies	gram positive cocci in groups,few pus cells	S.aureus	-	-	S	S	S	R
38	624123	Yellow	Sec	Infected eczema	gram positive cocci in cluster,many pus cells	S.aureus	-	-	S	S	S	S
39	858546	-	P	Impetigo contagiosa	gram positive cocci in cluster,many pus cells	S.aureus	-	-	S	S	S	S
40	892443	-	P	Folliculitis	many pus cells	no growth	-	-	-	-	-	-
41	859772	-	Sec	Infected eczema	gram positive cocci in cluster,few pus cells	S.aureus	-	-	S	S	S	S
42	245182	-	P	Abscess	gram positive cocci in groups,many pus cells	S.aureus	-	-	S	S	S	R
43	57891	-	P	Impetigo contagiosa	gram positive cocci in cluster,many pus cells	S.aureus	-	-	S	S	S	R
44	866147	-	Sec	Infected eczema	many pus cells	no growth	-	-	-	-	-	-
45	532997	-	Sec	Infected eczema	many pus cells	no growth	-	-	-	-	-	-
46	866792	-	Sec	Infected scabies	gram positive cocci in cluster,few pus cells	S.aureus	-	-	S	S	S	S
47	875738	-	Sec	Infected tinea	gram positive cocci in cluster,many pus cells	S.aureus	-	-	S	S	S	S
48	848220	-	P	Furuncle	gram negative bacilli,many pus cells	E.coli	-	-	R	MS	-	R
49	88971	-	P	Furuncle	gram negative bacilli,many pus cells	E.coli	-	-	R	R	-	R
50	428622	-	P	Impetigo contagiosa	gram positive cocci in cluster,many pus cells	S.aureus	-	-	S	S	S	R
51	911534	-	Sec	Infected scabies	gram positive cocci in groups,few pus cells	S.aureus	-	-	S	S	S	S
52	881931	-	P	Furuncle	gram positive cocci in groups,many pus cells	S.aureus	-	-	S	S	S	S
53	901862	-	P	Folliculitis	gram positive cocci in cluster,many pus cells	S.aureus	-	-	S	S	S	R
54	910036	-	P	Furuncle	gram positive cocci in groups,few pus cells	S.aureus	-	-	S	S	S	R
55	910035	-	P	Impetigo contagiosa	gram positive cocci in cluster,many pus cells	S.aureus	-	-	R	S	S	S
56	643229	Yellow	P	Furuncle	gram positive cocci in cluster,many pus cells	S.aureus	-	-	S	S	S	S
57	784587	-	P	Impetigo contagiosa	gram positive cocci in groups,few pus cells	S.aureus	-	-	S	S	S	S
58	866738	-	Sec	Infected eczema	gram positive cocci in groups,many pus cells	S.aureus	-	-	S	S	S	S
59	859772	-	Sec	Infected eczema	gram positive cocci in groups,many pus cells	S.aureus	-	-	S	S	S	R
60	201778	-	Sec	Infected scabies	gram positive cocci in groups,many pus cells	S.aureus	-	-	S	S	S	S
61	891931	-	P	Furuncle	gram positive cocci in groups,many pus cells	S.aureus	-	-	R	-	-	R
62	893020	-	P	Impetigo contagiosa	gram positive cocci in cluster,many pus cells	S.aureus	-	-	S	S	S	S
63	892443	-	P	Impetigo contagiosa	gram positive cocci in cluster,many pus cells	S.aureus	-	-	S	S	S	R
64	384767	-	Sec	Infected eczema	gram positive cocci in groups,few pus cells	S.aureus	-	-	S	S	S	S
65	905126	-	Sec	Infected eczema	gram positive cocci in groups,many pus cells	S.aureus	-	-	S	S	S	S
66	883401	-	Sec	Infected scabies	gram positive cocci in groups,many pus cells	S.aureus	-	-	S	S	S	S
67	211879	-	P	Impetigo contagiosa	gram positive cocci in cluster,few pus cells	S.aureus	-	-	S	S	S	S

S.No.	MRD No.	Antibiotic susceptibility													
		Clindamycin	Erythromycin	Gentamycin	Linezolid	Penicillin	Teicoplanin	Vancomycin	Cefotaxime	Amoxiclav	Cefoperazone + Sulbactam	Piperacillin	Piperacillin+Tazobactam	Oxacillin	Ticarcillin+clavulanic acid
32	839745	S	R	S	S	R	S	S	-	-	-	-	-	-	-
33	754276	S	S	S	S	S	S	S	-	-	-	-	-	-	-
34	71801	S	R	S	S	R	S	S	-	-	-	-	-	-	-
35	844596	S	S	S	S	S	S	S	-	-	-	-	-	-	-
36	864744	S	S	S	S	S	S	S	-	-	-	-	-	-	-
37	861854	S	S	R	S	R	S	S	-	-	-	-	-	-	-
38	624123	S	S	S	S	S	S	S	-	-	-	-	-	-	-
39	858546	S	R	S	S	R	S	S	-	-	-	-	-	-	-
40	892443	-	-	-	-	-	-	-	-	-	-	-	-	-	-
41	859772	S	S	S	S	S	S	S	-	-	-	-	-	-	-
42	245182	R	R	S	S	R	S	S	-	-	-	-	-	-	-
43	57891	S	R	R	S	R	S	S	-	-	-	-	-	-	-
44	866147	-	-	-	-	-	-	-	-	-	-	-	-	-	-
45	532997	-	-	-	-	-	-	-	-	-	-	-	-	-	-
46	866792	S	R	S	S	R	S	S	-	-	-	-	-	-	-
47	875738	S	S	S	S	S	S	S	-	-	-	-	-	-	-
48	848220	-	-	S	-	-	-	R	-	R	S	R	MS	-	-
49	88971	-	-	R	-	-	-	-	R	-	S	S	S	-	-
50	428622	S	R	R	S	R	S	S	-	-	-	-	-	-	-
51	911534	S	S	S	S	S	S	S	-	-	-	-	-	-	-
52	881931	S	S	S	S	S	S	S	-	-	-	-	-	-	-
53	901862	S	S	S	S	S	S	S	-	-	-	-	-	-	-
54	910036	R	R	S	S	R	S	S	-	-	-	-	-	-	-
55	910035	S	S	S	S	R	S	S	-	-	-	-	-	-	-
56	643229	S	S	S	S	R	S	S	-	-	-	-	-	-	-
57	784587	S	S	S	S	S	S	S	-	-	-	-	-	-	-
58	866738	S	R	S	S	R	S	S	-	-	-	-	-	-	-
59	859772	S	S	S	S	S	S	S	-	-	-	-	-	-	-
60	201778	S	S	S	S	S	S	S	-	-	-	-	-	-	-
61	891931	-	R	S	S	R	S	S	-	-	-	-	-	S	-
62	893020	S	S	S	S	S	S	S	-	-	-	-	-	-	-
63	892443	S	S	S	S	R	S	S	-	-	-	-	-	-	-
64	384767	S	S	S	S	S	S	S	-	-	-	-	-	-	-
65	905126	S	S	S	S	S	S	S	-	-	-	-	-	-	-
66	883401	S	S	S	S	S	S	S	-	-	-	-	-	-	-
67	211879	S	S	S	S	S	S	S	-	-	-	-	-	-	-

S.No.	MRD No.	Name	Age (years)	Sex	OPD/ IPD	Clinical presentation			Risk factors			Symptoms			Morphology of lesions			
						Duration of Disease (days)	Site	Size (cm)	Poor hygiene	H/o Diabetes	Immunosuppression	Redness	Pain	Swelling	Erythema	Crusting	Crusting (color)	Oozing
68	698735	PK	6	F	OPD	2	Head and neck	0.4	N	N	N	Y	Y	N	Y	Y	Honey colored	N
69	659570	SK	2	F	OPD	1	Head and neck	0.4	Y	N	N	Y	Y	N	Y	Y	Honey colored	N
70	230791	CK	5	F	OPD	1	Head and neck	0.4	Y	N	N	Y	Y	N	Y	Y	Honey colored	N
71	885055	PP	7	M	OPD	6	Head and neck	0.3	Y	N	N	Y	N	N	Y	N	-	N
72	828862	AS	3	F	OPD	3	Head and neck	0.3	Y	N	N	Y	Y	Y	Y	N	-	N
73	844541	S	6	F	OPD	2	Head and neck	0.4	Y	N	N	Y	Y	N	Y	Y	Honey colored	N
74	127112	MK	11	F	OPD	2	Upper limb	0.5	Y	N	N	Y	Y	N	Y	Y	Honey colored	N
75	856634	L	15	M	OPD	6	Head and neck	1	Y	N	N	N	N	N	N	N	-	N
76	686361	KK	4	F	OPD	1	Head and neck	1	Y	N	N	Y	Y	N	Y	Y	Honey colored	N
77	642956	S	6	F	OPD	3	Trunk	1	Y	N	N	Y	Y	N	Y	Y	Honey colored	N
78	866204	HT	14	M	OPD	6	Trunk	2	Y	N	N	N	N	N	N	N	-	N
79	867832	JM	5	M	OPD	2	Head and neck	1	N	N	N	Y	Y	N	Y	Y	Honey colored	N
80	920302	A	3	M	OPD	1	Head and neck	0.5	N	N	N	Y	Y	N	Y	Y	Honey colored	N
81	920092	KS	1	M	OPD	1	Head and neck	0.4	N	N	N	Y	Y	N	Y	Y	Honey colored	N
82	909518	M	1	M	OPD	1	Head and neck	0.5	N	N	N	Y	Y	N	Y	Y	Honey colored	N
83	919690	M	1	M	OPD	9	Head and neck	0.5	Y	N	N	N	N	N	N	Y	Reddish-yellow	N
84	782122	L	3	F	OPD	1	Head and neck	0.3	N	N	N	Y	Y	N	Y	Y	Honey colored	N
85	828613	M	6	M	OPD	3	Head and neck	0.4	N	N	N	Y	Y	N	Y	Y	Honey colored	N
86	21439	R	8	F	OPD	6	Head and neck	0.4	Y	N	N	N	N	N	N	N	-	N
87	517006	AN	4	F	OPD	2	Head and neck	0.3	Y	N	N	Y	Y	N	Y	Y	Honey colored	Y
88	912792	E	6	F	OPD	2	Head and neck	0.5	Y	N	N	Y	Y	N	Y	Y	Honey colored	Y
89	905144	YK	11	F	OPD	6	Buttock	1	Y	N	N	N	N	N	N	Y	Reddish-yellow	N
90	908704	AS	5	M	OPD	4	Buttock	0.4	Y	N	N	Y	Y	N	Y	N	-	N
91	910929	K	3	F	OPD	2	Perioral	0.5	Y	N	N	Y	Y	N	Y	Y	Honey colored	Y
92	910214	B	4	M	OPD	2	Perioral	0.5	Y	N	N	Y	Y	N	Y	Y	Honey colored	Y
93	895566	S	2	M	OPD	3	Perioral	0.4	Y	N	N	Y	Y	N	Y	Y	Honey colored	Y
94	905764	N	7	F	OPD	2	Perioral	0.5	Y	N	N	Y	Y	N	Y	Y	Honey colored	Y

S.No.	MRD No.	Oozing (color)	Type of pyoderma	Clinical Diagnosis	Gram's staining	Identification		MRSA	Cotrimoxazole	Amikacin	Cefoxitin	Ciprofloxacin
						organism	organism					
68	698735	-	P	Impetigo contagiosa	gram positive cocci in cluster,many pus cells	S.aureus	-	-	S	S	S	S
69	659570	-	P	Impetigo contagiosa	gram positive cocci in cluster,many pus cells	S.aureus	-	-	S	S	S	S
70	230791	-	P	Impetigo contagiosa	gram positive cocci in cluster,few pus cells	S.aureus	-	-	S	S	S	S
71	885055	-	Sec	Infected tinea	gram positive cocci in cluster,many pus cells	S.aureus	-	-	S	S	S	S
72	828862	-	P	Folliculitis	gram positive cocci in cluster,many pus cells	S.aureus	-	Y	S	S	R	R
73	844541	-	P	Impetigo contagiosa	gram positive cocci in cluster,few pus cells	S.aureus	-	-	S	S	S	S
74	127112	-	P	Folliculitis	gram positive cocci in cluster,many pus cells	S.aureus	-	Y	S	S	R	R
75	856634	-	Sec	Infected scabies	gram positive cocci in groups,many pus cells	S.aureus	-	-	S	S	S	S
76	686361	-	P	Impetigo contagiosa	gram positive cocci in cluster,many pus cells	S.aureus	-	-	S	S	S	R
77	642956	-	P	Impetigo contagiosa	gram positive cocci in cluster,few pus cells	S.aureus	-	-	S	S	S	S
78	866204	-	Sec	Infected scabies	gram positive cocci in groups,many pus cells	S.aureus	-	-	S	S	S	S
79	867832	-	P	Impetigo contagiosa	gram positive cocci in cluster,many pus cells	S.aureus	-	-	S	S	S	S
80	920302	-	P	Impetigo contagiosa	gram positive cocci in cluster,many pus cells	S.aureus	-	-	S	S	S	S
81	920092	-	P	Impetigo contagiosa	gram positive cocci in cluster,few pus cells	S.aureus	-	-	S	S	S	S
82	909518	-	P	Impetigo contagiosa	gram positive cocci in cluster,many pus cells	S.aureus	-	-	S	S	S	R
83	919690	-	P	Folliculitis	gram positive cocci in cluster,many pus cells	S.aureus	-	-	S	S	S	R
84	782122	-	P	Impetigo contagiosa	gram positive cocci in cluster,few pus cells	S.aureus	-	-	S	S	S	S
85	828613	-	P	Impetigo contagiosa	gram positive cocci in cluster,few pus cells	S.aureus	-	-	S	S	S	S
86	21439	-	Sec	Infected scabies	gram positive cocci in groups,many pus cells	S.aureus	-	-	S	S	S	S
87	517006	Yellow	P	Impetigo contagiosa	gram positive cocci in cluster,many pus cells	S.aureus	-	-	S	S	S	S
88	912792	Yellow	P	Impetigo contagiosa	gram positive cocci in cluster,many pus cells	S.aureus	-	-	S	S	S	S
89	905144	-	P	Folliculitis	gram positive cocci in cluster,few pus cells	S.aureus	-	-	S	S	S	R
90	908704	-	P	Furuncle	gram positive cocci in groups,few pus cells	S.aureus	-	-	S	S	S	S
91	910929	Yellow	P	Impetigo contagiosa	gram positive cocci in cluster,few pus cells	S.aureus	-	-	S	S	S	S
92	910214	Yellow	P	Impetigo contagiosa	gram positive cocci in cluster,few pus cells	S.aureus	-	-	S	S	S	S
93	895566	Yellow	P	Impetigo contagiosa	gram positive cocci in cluster,many pus cells	S.aureus	-	-	S	S	S	S
94	905764	Yellow	P	Impetigo contagiosa	gram positive cocci in cluster,few pus cells	S.aureus	-	-	S	S	S	S

S.No.	MRD No.	Antibiotic susceptibility													
		Clindamycin	Erythromycin	Gentamycin	Linezolid	Penicillin	Teicoplanin	Vancomycin	Cefotaxime	Amoxiclav	Cefoperazone + Sulbactam	Piperacillin	Piperacillin+Tazobactam	Oxacillin	Ticarcillin+clavulanic acid
68	698735	S	S	S	S	S	S	S	-	-	-	-	-	-	-
69	659570	S	S	S	S	S	S	S	-	-	-	-	-	-	-
70	230791	S	S	S	S	S	S	S	-	-	-	-	-	-	-
71	885055	S	S	S	S	S	S	S	-	-	-	-	-	-	-
72	828862	S	R	S	S	R	R	S	-	-	-	-	-	-	-
73	844541	S	S	S	S	S	S	S	-	-	-	-	-	-	-
74	127112	S	R	S	S	R	R	S	-	-	-	-	-	-	-
75	856634	S	S	S	S	S	S	S	-	-	-	-	-	-	-
76	686361	S	R	R	S	R	S	S	-	-	-	-	-	-	-
77	642956	S	S	S	S	S	S	S	-	-	-	-	-	-	-
78	866204	S	S	S	S	S	S	S	-	-	-	-	-	-	-
79	867832	S	S	S	S	S	S	S	-	-	-	-	-	-	-
80	920302	S	S	S	S	S	S	S	-	-	-	-	-	-	-
81	920092	S	S	S	S	S	S	S	-	-	-	-	-	-	-
82	909518	S	S	S	S	R	S	S	-	-	-	-	-	-	-
83	919690	S	S	S	S	S	S	S	-	-	-	-	-	-	-
84	782122	S	S	S	S	S	S	S	-	-	-	-	-	-	-
85	828613	S	S	S	S	S	S	S	-	-	-	-	-	-	-
86	21439	S	S	S	S	S	S	S	-	-	-	-	-	-	-
87	517006	S	S	S	S	S	S	S	-	-	-	-	-	-	-
88	912792	S	S	S	S	S	S	S	-	-	-	-	-	-	-
89	905144	S	S	S	S	S	S	S	-	-	-	-	-	-	-
90	908704	S	S	S	S	S	S	S	-	-	-	-	-	-	-
91	910929	S	S	S	S	S	S	S	-	-	-	-	-	-	-
92	910214	S	S	S	S	S	S	S	-	-	-	-	-	-	-
93	895566	S	S	S	S	S	S	S	-	-	-	-	-	-	-
94	905764	S	S	S	S	S	S	S	-	-	-	-	-	-	-

						Clinical presentation			Risk factors			Symptoms			Morphology of lesions			
S.No.	MRD No.	Name	Age (years)	Sex	OPD/ IPD	Duration of Disease (days)	Site	Size (cm)	Poor hygiene	H/o Diabetes	Immunosuppression	Redness	Pain	Swelling	Erythema	Crusting	Crusting (color)	Oozing
95	199963	A	3	F	OPD	3	Perioral	0.3	Y	N	N	Y	Y	N	Y	Y	Honey colored	Y
96	899995	HD	3	M	OPD	2	Perioral	0.4	Y	N	N	Y	Y	N	Y	Y	Honey colored	Y
97	500882	H	10	F	OPD	1	Perioral	1	Y	N	N	Y	Y	N	Y	Y	Honey colored	Y
98	896311	Z	7	M	OPD	3	Perioral	0.5	Y	N	N	Y	Y	N	Y	Y	Honey colored	Y
99	899112	IS	5	M	OPD	3	Perioral	0.5	Y	N	N	Y	Y	N	Y	Y	Honey colored	Y
100	895456	U	6	M	OPD	2	Perioral	0.5	Y	N	N	Y	Y	N	Y	Y	Honey colored	Y

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S.No.	MRD No.	Oozing (color)	Type of pyoderma	Clinical Diagnosis	Gram's staining	Identification		MRSA				
						organism	organism		Cotrimoxazole	Amikacin	Cefoxitin	Ciprofloxacin
95	199963	Yellow	P	Impetigo contagiosa	gram positive cocci in cluster,many pus cells	S.aureus	-	-	S	S	S	S
96	899995	Yellow	P	Impetigo contagiosa	gram positive cocci in cluster,many pus cells	S.aureus	-	-	S	S	S	R
97	500882	Yellow	P	Impetigo contagiosa	gram positive cocci in cluster,few pus cells	S.aureus	-	-	S	S	S	S
98	896311	Yellow	P	Impetigo contagiosa	gram positive cocci in cluster,few pus cells	S.aureus	-	-	S	S	S	S
99	899112	Yellow	P	Impetigo contagiosa	gram positive cocci in cluster,many pus cells	S.aureus	-	-	S	S	S	S
100	895456	Yellow	P	Impetigo contagiosa	gram positive cocci in cluster,few pus cells	S.aureus	-	-	S	S	S	S

S.No.	MRD No.	Antibiotic susceptibility													
		Clindamycin	Erythromycin	Gentamycin	Linezolid	Penicillin	Teicoplanin	Vancomycin	Cefotaxime	Amoxiclav	Cefoperazone + Sulbactam	Piperacillin	Piperacillin+Tazobactam	Oxacillin	Ticarcillin+clavulanic acid
95	199963	S	S	S	S	S	S	S	-	-	-	-	-	-	-
96	899995	S	S	S	S	S	S	S	-	-	-	-	-	-	-
97	500882	S	S	S	S	S	S	S	-	-	-	-	-	-	-
98	896311	S	S	S	S	S	S	S	-	-	-	-	-	-	-
99	899112	S	S	S	S	S	S	S	-	-	-	-	-	-	-
100	895456	S	R	S	S	R	S	S	-	-	-	-	-	-	-

						Clinical presentation			Risk factors				Symptoms			Morphology of lesions		
S.No.	MRD No.	Name	Age (years)	Sex	OPD/ IPD	Duration of Disease (days)	Site	Size (cm)	Poor hygiene	H/o Diabetes	Duration of diabetes (years)	Immunosuppression	Redness	Pain	Swelling	Erythema	Crusting	Crusting (color)
1	628010	SK	49	F	OPD	3	Lower limb	1	Y	Y	7	N	Y	Y	Y	Y	Y	brownish-red
2	637630	GS	26	M	OPD	5	Trunk	2	N	N	-	Y	Y	Y	Y	Y	Y	brownish-red
3	902328	RA	35	M	OPD	10	Trunk	2	N	N	-	N	Y	Y	Y	Y	N	-
4	632989	NK	33	F	OPD	2	Lower limb	1	N	N	-	N	Y	Y	Y	N	N	yellowish-brown
5	612570	M	23	F	OPD	2	Upper limb	1	N	N	-	N	Y	Y	Y	N	N	-
6	454044	ML	60	M	OPD	1	Lower limb	1	N	N	-	N	Y	Y	Y	N	N	-
7	654789	MK	30	F	IPD	10	Lower limb	1	N	N	-	N	Y	Y	N	N	N	-
8	658768	RS	46	M	IPD	3	Trunk	3	N	Y	5	N	Y	Y	Y	N	Y	-
9	112348	RB	32	M	OPD	6	Head and neck	4	Y	N	-	N	N	Y	N	Y	N	dirty brown
10	278413	V	35	F	OPD	12	Trunk	4	N	N	-	N	Y	Y	Y	N	N	-
11	161326	NS	54	M	OPD	7	Lower limb	1	Y	Y	3	N	Y	N	N	Y	Y	dirty brown
12	696558	VP	48	F	IPD	7	Upper limb	2	N	Y	3	N	N	Y	N	N	Y	-
13	645396	KD	47	M	OPD	3	Head and neck	0.3	Y	N	-	N	Y	Y	Y	Y	N	dirty brown
14	657919	VK	32	M	IPD	14	Trunk	0.5	N	N	-	N	Y	Y	Y	N	N	-
15	653399	SS	65	M	OPD	5	Trunk	1	N	N	-	N	Y	Y	Y	N	N	-
16	693340	RK	60	F	IPD	7	Trunk	6	Y	Y	1	Y	Y	Y	Y	Y	Y	-
17	641538	GG	38	F	OPD	8	Upper limb	2	N	N	-	N	Y	Y	Y	N	N	-
18	735655	KK	29	F	OPD	3	Trunk	0.5	N	N	-	N	Y	Y	Y	N	N	-
19	737882	CM	56	M	OPD	3	Trunk	2	N	N	-	N	Y	Y	Y	N	N	-
20	750904	MK	42	F	OPD	6	Trunk	1	Y	N	-	N	Y	Y	Y	Y	N	-
21	712804	IR	32	F	OPD	5	Genitalia	0.5	Y	N	-	N	Y	N	N	Y	N	-
22	732227	PK	42	F	OPD	4	Head and neck	2	N	N	-	N	N	Y	N	N	N	dirty brown
23	195936	HS	28	M	OPD	7	Trunk	4	N	N	-	N	Y	Y	Y	N	N	dirty brown
24	664771	S	62	M	OPD	6	Lower limb	2	Y	N	-	N	N	N	N	Y	N	-
25	564248	VC	65	F	IPD	10	Lower limb	6	Y	Y	20	Y	Y	Y	Y	Y	Y	-
26	780973	MS	27	M	OPD	10	Lower limb	2	N	N	-	N	N	Y	N	N	N	-
27	223178	SC	73	M	OPD	14	Lower limb	2	N	Y	20	N	Y	Y	N	N	Y	dirty brown
28	842293	R	46	M	OPD	3	Head and neck	0.3	N	Y	15	N	Y	Y	N	N	Y	dirty brown
29	805059	GY	27	M	OPD	6	Lower limb	3	Y	N	-	N	Y	Y	N	Y	N	yellowish-brown
30	610471	DS	55	M	IPD	12	Lower limb	4	Y	N	-	N	Y	Y	Y	Y	N	-
31	682797	AS	70	M	IPD	7	Genitalia	1	N	Y	35	N	Y	Y	Y	N	Y	-
32	447047	BS	23	M	IPD	5	Trunk	2	N	N	-	N	Y	Y	Y	N	N	-
33	722480	KR	28	M	OPD	5	Lower limb	3	Y	N	-	N	Y	Y	Y	Y	N	yellowish-brown
34	483132	J	50	F	IPD	6	Lower limb	5	Y	N	-	Y	N	N	N	Y	N	-
35	45668	JS	43	M	OPD	3	Trunk	0.5	Y	Y	7	N	Y	Y	N	Y	Y	-
36	787333	PK	53	F	OPD	3	Trunk	2	N	Y	8	N	N	Y	N	N	Y	dirty brown
37	814131	RV	66	M	OPD	5	Head and neck	0.7	N	Y	6	N	Y	Y	Y	N	Y	yellowish-brown
38	170431	GK	36	F	OPD	5	Trunk	0.3	Y	N	-	N	N	Y	N	Y	N	-

S.No.	MRD No.	s		Type of pyoderma	Clinical Diagnosis	Gram's staining	Identification		MRSA				
		Oozing	Oozing (color)				organism	organism		Cotrimoxazole	Amikacin	Cefoxitin	Ciprofloxacin
1	628010	Y	yellowish	P	Abscess	gram positive cocci in cluster	S.aureus	-	-	S	S	S	MS
2	637630	Y	yellowish	P	Furuncle	gram positive cocci in cluster,few pus cells	S.aureus	-	-	S	S	S	S
3	902328	Y	yellowish	P	Carbuncle	gram positive cocci in groups,gram negative bacilli	S.aureus	E.coli	Y	R	S	-	R
4	632989	N	-	P	Folliculitis	gram positive cocci in cluster,many pus cells	S.aureus	-	-	S	S	S	R
5	612570	N	-	P	Furuncle	few pus cells	no growth	-	-	-	-	-	-
6	454044	Y	yellowish	P	DCPA	gram positive cocci in cluster,few pus cells	S.aureus	-	-	S	S	S	S
7	654789	N	-	P	Furuncle	gram positive cocci in groups,gram negative bacilli	S.aureus	P.aeruginosa	Y	S	S	-	R
8	658768	N	-	P	Abscess	gram positive cocci in cluster,few pus cells	S.aureus	-	Y	R	-	-	R
9	112348	N	-	P	Ecthyma	gram negative bacilli,few pus cells	E.coli	-	-	S	S	-	S
10	278413	N	-	P	Abscess	gram positive cocci in cluster,few pus cells	S.aureus	-	Y	S	S	R	R
11	161326	N	-	Sec	Infected tinea	gram negative bacilli,many pus cells	P.aeruginosa	citrobacter	-	S	S	-	S
12	696558	Y	yellowish	Sec	Vasculitic ulcer	gram negative bacilli,many pus cells	Proteus vulgaris	-	-	R	S	-	S
13	645396	N	-	P	Sycosis barbae	gram positive cocci in cluster,many pus cells	S.aureus	-	-	S	S	S	R
14	657919	Y	yellowish	P	Furuncle	gram positive cocci in cluster,many pus cells	S.aureus	-	-	S	S	-	R
15	653399	Y	yellowish	P	Furuncle	gram positive cocci in cluster,few pus cells	S.aureus	-	-	S	S	S	S
16	693340	N	-	P	Carbuncle	gram positive cocci in groups,gram negative bacilli	S.aureus	E.coli	Y	R	S	-	R
17	641538	N	-	P	Furuncle	gram positive cocci in cluster,many pus cells	S.aureus	-	-	S	S	S	R
18	735655	N	-	P	Furuncle	gram positive cocci in cluster,many pus cells	S.aureus	-	-	R	S	S	R
19	737882	N	-	P	Abscess	gram positive cocci in cluster,many pus cells	S.aureus	-	-	MS	S	S	MS
20	750904	N	-	Sec	Infected tinea	gram positive cocci in cluster,many pus cells	S.aureus	-	-	R	S	S	R
21	712804	N	-	P	Folliculitis	gram positive cocci in cluster,few pus cells	S.aureus	-	-	S	S	S	S
22	732227	Y	yellowish	P	Furuncle	gram positive cocci in cluster,few pus cells	Enterococcus	-	-	-	-	-	S
23	195936	Y	Reddish-yellow	P	Abscess	gram positive cocci in cluster,many pus cells	S.aureus	-	-	S	S	S	R
24	664771	Y	yellowish	Sec	Infected tinea	gram negative bacilli,many pus cells	P.aeruginosa	-	-	-	S	-	S
25	564248	N	-	P	Cellulitis	gram negative bacilli,many pus cells	P.aeruginosa	-	-	-	S	-	R
26	780973	N	-	Sec	Infected eczema	gram positive cocci in cluster,few pus cells	S.aureus	P.aeruginosa	-	S	S	S	S
27	223178	N	-	Sec	Vasculitic ulcer	gram negative bacilli,many pus cells	P.aeruginosa	-	-	S	S	-	S
28	842293	N	-	P	Sycosis barbae	gram positive cocci in cluster,few pus cells	S.aureus	-	-	S	S	S	R
29	805059	N	-	Sec	Infected LSC	gram positive cocci in cluster,few pus cells	S.aureus	Enterobacter	-	S	S	S	S
30	610471	Y	Reddish-yellow	P	Cellulitis	gram negative bacilli,few pus cells	Enterobacter	citrobacter	-	S	S	-	S
31	682797	Y	yellowish	Sec	Infected tinea	gram negative bacilli,few pus cells	K.pneumoniae	-	-	S	S	-	MS
32	447047	Y	yellowish	P	Abscess	gram positive cocci in cluster,few pus cells	S.aureus	-	-	R	S	S	S
33	722480	N	-	Sec	Infected eczema	gram positive cocci in cluster,few pus cells	S.aureus	-	-	S	S	S	S
34	483132	Y	yellowish	P	Carbuncle	gram positive cocci in cluster,few pus cells	S.aureus	-	-	S	S	S	MS
35	45668	N	-	P	Furuncle	gram negative bacilli,many pus cells	P.aeruginosa	-	-	-	S	-	S
36	787333	N	-	Sec	Infected eczema	Many pus cells	no growth	-	-	-	-	-	-
37	814131	Y	Reddish-yellow	P	Furuncle	gram positive cocci in cluster,many pus cells	S.aureus	-	-	S	S	S	R
38	170431	N	-	Sec	Infected tinea	gram positive cocci in cluster,few pus cells	S.hemolyticus	-	-	R	-	-	R

S.No.	MRD No.	Antibiotic susceptibility													
		Clindamycin	Erythromycin	Gentamycin	Linezolid	Penicillin	Teicoplanin	Vancomycin	Cefotaxime	Amoxiclav	Cefoperazone+ Sulbactam	Piperacillin	Piperacillin+Tazobactam	Oxacillin	Ticarcillin+clavulanic acid
1	628010	S	S	S	S	S	S	S	-	-	-	-	-	-	-
2	637630	S	S	S	S	S	S	S	-	-	-	-	-	-	-
3	902328	S	R	R	S	S	S	S	-	R	R	-	R	R	-
4	632989	S	S	S	S	R	S	S	-	-	-	-	-	-	-
5	612570	-	-	-	-	-	-	-	-	-	-	-	-	-	-
6	454044	S	S	-	S	S	S	S	-	-	-	-	-	-	-
7	654789	S	S	MS	S	-	S	S	-	R	R	-	S	R	MS
8	658768	S	R	S	S	R	S	S	-	-	-	-	-	R	-
9	112348	-	-	S	-	R	-	-	S	-	S	S	S	-	-
10	278413	S	S	S	S	R	S	S	-	-	-	-	-	-	-
11	161326	-	-	S	-	R	-	-	S	-	S	S	S	-	-
12	696558	-	-	S	-	R	-	-	S	-	S	-	S	-	-
13	645396	R	R	S	S	R	S	S	-	-	-	-	-	-	-
14	657919	S	S	S	S	R	S	S	-	-	-	-	-	-	-
15	653399	S	R	S	S	R	S	S	-	-	-	-	-	-	-
16	693340	S	R	R	S	S	S	S	-	R	R	-	R	R	-
17	641538	S	S	MS	S	R	S	S	-	-	-	-	-	-	-
18	735655	S	S	S	S	R	S	S	-	-	-	-	-	-	-
19	737882	S	S	S	S	R	S	S	-	-	-	-	-	-	-
20	750904	S	MS	S	S	R	S	S	-	-	-	-	-	-	-
21	712804	S	S	S	S	R	S	S	-	-	-	-	-	-	-
22	732227	-	S	S	S	S	S	S	-	S	-	-	-	-	-
23	195936	S	R	S	S	R	S	S	-	-	-	-	-	-	-
24	664771	-	-	S	-	-	-	-	-	-	S	S	S	-	-
25	564248	-	-	S	-	-	-	-	-	-	S	S	S	-	-
26	780973	S	S	S	S	R	S	S	-	-	S	S	S	-	-
27	223178	-	-	S	-	-	-	-	-	-	S	S	S	-	-
28	842293	S	R	S	S	R	S	S	-	-	S	S	S	-	-
29	805059	S	S	S	S	R	S	S	S	-	-	-	-	-	-
30	610471	-	-	S	-	-	-	-	-	R	S	-	S	-	-
31	682797	-	-	S	-	-	-	-	-	S	S	-	MS	-	-
32	447047	R	R	S	S	R	S	S	-	-	-	-	-	-	-
33	722480	S	S	R	S	R	S	S	-	-	-	-	-	-	-
34	483132	S	S	S	S	S	S	S	-	-	-	-	-	-	-
35	45668	-	-	S	-	-	-	-	-	-	S	S	S	-	-
36	787333	-	-	-	-	-	-	-	-	-	-	-	-	-	-
37	814131	S	S	S	S	R	S	S	-	-	-	-	-	-	-
38	170431	R	R	R	S	R	-	-	-	-	-	-	-	-	-

						Clinical presentation			Risk factors				Symptoms			Morphology of lesions		
S.No.	MRD No.	Name	Age (years)	Sex	OPD/ IPD	Duration of Disease (days)	Site	Size (cm)	Poor hygiene	H/o Diabetes	Duration of diabetes (years)	Immunosuppression	Redness	Pain	Swelling	Erythema	Crusting	Crusting (color)
39	515887	RG	33	F	OPD	6	Upper limb	2	N	N	-	N	Y	Y	Y	N	N	-
40	234134	ZS	73	M	OPD	3	Lower limb	0.2	N	Y	26	N	Y	Y	N	N	Y	-
41	732874	NK	30	F	OPD	2	Head and neck	0.3	N	N	-	N	Y	Y	N	N	N	-
42	658831	CL	58	M	OPD	10	Lower limb	2	N	N	-	N	N	Y	N	N	N	dirty brown
43	690120	GS	41	M	OPD	4	Lower limb	5	N	N	-	N	Y	Y	Y	N	N	-
44	415774	RG	46	F	OPD	10	Trunk	2	N	N	-	N	Y	Y	Y	N	N	-
45	68755	BR	68	M	OPD	4	Head and neck	2	N	Y	4	N	Y	Y	N	N	Y	-
46	182941	M	32	F	OPD	3	Trunk	3	N	N	-	N	Y	Y	Y	N	N	-
47	664001	AM	55	F	OPD	8	Lower limb	0.5	Y	N	-	N	Y	Y	Y	Y	N	-
48	720987	R	53	F	OPD	2	Lower limb	0.5	N	N	-	N	Y	Y	N	N	N	-
49	532714	N	38	F	OPD	5	Lower limb	0.3	N	Y	10	N	Y	Y	Y	N	Y	-
50	433260	KK	51	M	OPD	4	Upper limb	0.4	N	N	-	N	Y	Y	Y	N	N	-
51	738937	HS	22	M	OPD	3	Lower limb	1	N	N	-	N	Y	Y	N	N	N	-
52	657324	BK	45	F	OPD	10	Lower limb	0.2	N	N	-	N	Y	Y	Y	N	N	-
53	744113	JK	38	M	OPD	4	Trunk	2	Y	N	-	Y	N	Y	N	Y	N	-
54	723931	RM	57	M	OPD	7	Trunk	3	N	Y	19	N	Y	Y	Y	N	Y	dirty brown
55	649564	JG	24	F	OPD	3	Head and neck	0.3	N	N	-	N	Y	Y	N	N	N	-
56	742257	NK	56	F	OPD	3	Trunk	1	Y	Y	8	Y	Y	Y	N	Y	Y	-
57	812507	R	25	M	OPD	1	Head and neck	0.2	N	N	-	N	Y	Y	N	N	N	-
58	496983	R	41	F	OPD	6	Trunk	2	Y	Y	3	N	Y	Y	Y	Y	Y	-
59	808847	ZS	60	M	OPD	5	Trunk	2	Y	Y	26	N	Y	Y	Y	Y	Y	-
60	824858	CK	28	M	OPD	7	Lower limb	2	Y	N	-	N	N	N	N	Y	N	dirty brown
61	480078	RS	86	M	OPD	10	Upper limb	0.5	Y	Y	20	Y	Y	Y	N	Y	Y	dirty brown
62	828342	RK	51	M	OPD	7	Lower limb	4	N	N	-	N	Y	Y	Y	N	N	yellowish-brown
63	616387	BS	28	M	OPD	3	Trunk	0.2	N	N	-	N	N	Y	N	N	N	-
64	125277	S	29	F	OPD	15	Trunk	0.4	N	N	-	N	N	Y	N	N	N	-
65	827817	JS	46	M	OPD	4	Trunk	2	Y	Y	3	N	Y	Y	Y	Y	Y	-
66	838618	JS	75	M	IPD	7	Lower limb	6	N	Y	15	N	Y	Y	Y	N	Y	yellowish-brown
67	824812	KB	68	F	OPD	12	Trunk	2	Y	Y	17	N	Y	Y	Y	Y	Y	-
68	841254	SK	46	M	OPD	11	Trunk	3	Y	Y	3	N	Y	Y	Y	Y	Y	-
69	704636	LM	34	M	OPD	8	Head and neck	0.3	N	N	-	N	N	Y	N	N	N	-
70	841638	PK	46	F	OPD	5	Lower limb	1	Y	N	-	Y	N	N	N	Y	N	yellowish-brown
71	568701	KS	65	M	OPD	3	Trunk	0.4	N	Y	10	n	Y	Y	Y	N	Y	-
72	842668	PK	28	M	OPD	3	Trunk	2	N	N	-	N	Y	Y	Y	N	N	-
73	647137	RG	55	F	OPD	4	Trunk	0.2	N	N	-	N	N	Y	N	N	N	-
74	767943	PK	42	F	OPD	6	Lower limb	2	Y	N	-	N	Y	Y	N	Y	N	-
75	844837	HK	52	F	OPD	5	Trunk	0.6	Y	Y	7	N	Y	Y	Y	Y	Y	-

S.No.	MRD No.	s		Type of pyoderma	Clinical Diagnosis	Gram's staining	Identification		MRSA				
		Oozing	Oozing (color)				organism	organism		Cotrimoxazole	Amikacin	Cefoxitin	Ciprofloxacin
39	515887	N	-	P	Abscess	Many pus cells	no growth	-	-	-	-	-	-
40	234134	N	-	P	Folliculitis	gram positive cocci in cluster,many pus cells	S.aureus	-	-	S	S	S	R
41	732874	N	-	P	Folliculitis	gram positive cocci in cluster,many pus cells	S.aureus	-	-	MS	S	S	MS
42	658831	Y	Reddish-yellow	P	Ecthyma	gram negative bacilli,few pus cells	E.coli	-	-	S	S	-	S
43	690120	N	-	P	Cellulitis	few pus cells	no growth	-	-	-	-	-	-
44	415774	Y	yellowish	P	Folliculitis	Many pus cells	no growth	-	-	-	-	-	-
45	68755	N	-	P	Folliculitis	gram positive cocci in cluster,few pus cells	S.aureus	-	-	S	S	S	S
46	182941	Y	yellowish	P	Abscess	gram positive cocci in cluster	S.aureus	-	-	S	S	S	MS
47	664001	Y	yellowish	P	Folliculitis	few pus cells	no growth	-	-	-	-	-	-
48	720987	N	-	P	Folliculitis	few pus cells	no growth	-	-	-	-	-	-
49	532714	N	-	P	Acute paronychia	gram positive cocci in cluster,few pus cells	S.aureus	-	-	S	S	S	S
50	433260	N	-	P	Folliculitis	gram positive cocci in cluster,few pus cells	S.aureus	-	-	MS	S	S	R
51	738937	N	-	P	Folliculitis	gram positive cocci in cluster,many pus cells	S.aureus	-	-	S	S	S	R
52	657324	Y	yellowish	Sec	Infected LP	gram negative bacilli,few pus cells	E.coli	-	-	S	S	-	S
53	744113	Y	yellowish	Sec	Infected tinea	gram positive cocci in cluster,many pus cells	S.aureus	-	-	R	S	S	R
54	723931	Y	yellowish	P	Carbuncle	Many pus cells	no growth	-	-	-	-	-	-
55	649564	N	-	P	Folliculitis	few pus cells	no growth	-	-	-	-	-	-
56	742257	N	-	Sec	Infected tinea	Many pus cells	no growth	-	-	-	-	-	-
57	812507	N	-	P	Folliculitis	gram positive cocci in cluster,few pus cells	S.aureus	-	-	S	S	S	S
58	496983	N	-	P	Abscess	gram positive cocci in cluster	S.aureus	-	-	S	S	S	MS
59	808847	N	-	P	Abscess	gram positive cocci in clumps,many pus cells	S.aureus	-	-	R	S	S	R
60	824858	Y	yellowish	Sec	Infected eczema	Many pus cells	no growth	-	-	-	-	-	-
61	480078	N	-	Sec	Infected eczema	Many pus cells	no growth	-	-	-	-	-	-
62	828342	Y	Reddish-yellow	P	Cellulitis	gram positive cocci in groups,gram negative bacilli	S.aureus	E.coli	Y	R	S	-	R
63	616387	N	-	P	Folliculitis	gram positive cocci in cluster,few pus cells	S.aureus	-	-	S	S	S	S
64	125277	N	-	P	Folliculitis	gram positive cocci in cluster,few pus cells	S.aureus	-	-	S	S	S	S
65	827817	N	-	Sec	Infected tinea	gram positive cocci in groups,gram negative bacilli	S.aureus	-	-	R	S	-	R
66	838618	Y	Reddish-yellow	P	Cellulitis	Many pus cells	no growth	-	-	-	-	-	-
67	824812	Y	Reddish-yellow	P	Abscess	gram positive cocci in clumps,many pus cells	S.aureus	E.coli	-	S	S	S	R
68	841254	Y	Reddish-yellow	P	Abscess	gram positive cocci in clumps,few pus cells	S.aureus	-	-	S	S	S	S
69	704636	N	-	P	Folliculitis	gram positive cocci in cluster,few pus cells	S.aureus	-	-	S	S	S	S
70	841638	N	-	Sec	Infected tinea	gram negative bacilli,many pus cells	P.aeruginosa	-	-	-	S	-	S
71	568701	N	-	P	Folliculitis	few pus cells	no growth	-	-	-	-	-	-
72	842668	Y	Reddish-yellow	P	Abscess	gram positive cocci in cluster	S.aureus	-	-	R	S	S	S
73	647137	N	-	P	Folliculitis	gram positive cocci in cluster,few pus cells	S.aureus	-	-	S	S	S	S
74	767943	N	-	Sec	Infected tinea	Many pus cells	no growth	-	-	-	-	-	-
75	844837	N	-	Sec	Infected tinea	gram negative bacilli,many pus cells	P.aeruginosa	-	-	-	S	-	S

S.No.	MRD No.	Antibiotic susceptibility													
		Clindamycin	Erythromycin	Gentamycin	Linezolid	Penicillin	Teicoplanin	Vancomycin	Cefotaxime	Amoxiclav	Cefoperazone+ Sulbactam	Piperacillin	Piperacillin+Tazobactam	Oxacillin	Ticarcillin+clavulanic acid
39	515887	-	-	-	-	-	-	-	-	-	-	-	-	-	-
40	234134	S	R	S	S	R	S	S	-	-	-	-	-	-	-
41	732874	S	MS	S	S	R	S	S	-	-	-	-	-	-	-
42	658831	-	-	S	-	R	-	-	S	-	S	S	S	-	-
43	690120	-	-	-	-	-	-	-	-	-	-	-	-	-	-
44	415774	-	-	-	-	-	-	-	-	-	-	-	-	-	-
45	68755	S	S	S	S	R	S	S	-	-	-	-	-	-	-
46	182941	S	S	S	S	S	S	S	-	-	-	-	-	-	-
47	664001	-	-	-	-	-	-	-	-	-	-	-	-	-	-
48	720987	-	-	-	-	-	-	-	-	-	-	-	-	-	-
49	532714	S	S	S	S	R	S	S	-	-	-	-	-	-	-
50	433260	S	S	S	S	R	S	S	-	-	-	-	-	-	-
51	738937	R	R	S	S	R	S	S	-	-	-	-	-	-	-
52	657324	-	-	S	-	R	-	-	S	-	S	S	S	-	-
53	744113	S	MS	S	S	R	S	S	-	-	-	-	-	-	-
54	723931	-	-	-	-	-	-	-	-	-	-	-	-	-	-
55	649564	-	-	-	-	-	-	-	-	-	-	-	-	-	-
56	742257	-	-	-	-	-	-	-	-	-	-	-	-	-	-
57	812507	S	S	S	S	R	S	S	-	-	-	-	-	-	-
58	496983	S	S	S	R	MS	S	S	-	-	-	-	-	-	-
59	808847	S	S	S	R	MS	S	S	-	-	-	-	-	-	-
60	824858	-	-	-	-	-	-	-	-	-	-	-	-	-	-
61	480078	-	-	-	-	-	-	-	-	-	-	-	-	-	-
62	828342	S	R	R	S	S	S	S	-	R	R	-	R	R	-
63	616387	S	S	S	S	R	S	S	-	-	-	-	-	-	-
64	125277	S	S	S	S	S	S	S	-	-	-	-	-	-	-
65	827817	S	R	R	R	R	S	S	-	R	S	-	R	S	-
66	838618	-	-	-	-	-	-	-	-	-	-	-	-	-	-
67	824812	S	S	S	R	S	S	S	-	-	-	-	-	-	-
68	841254	S	S	S	S	S	S	S	-	-	-	-	-	-	-
69	704636	S	S	S	S	R	S	S	-	-	-	-	-	-	-
70	841638	-	-	S	-	-	-	-	-	-	S	S	S	-	-
71	568701	-	-	-	-	-	-	-	-	-	-	-	-	-	-
72	842668	S	S	S	R	R	S	S	-	-	-	-	-	-	-
73	647137	S	S	S	S	S	S	S	-	-	-	-	-	-	-
74	767943	-	-	-	-	-	-	-	-	-	-	-	-	-	-
75	844837	-	-	S	-	-	-	-	-	-	S	S	S	-	-

S.No.	MRD No.	Name	Age (years)	Sex	OPD/ IPD	Clinical presentation			Risk factors				Symptoms			Morphology of lesions		
						Duration of Disease (days)	Site	Size (cm)	Poor hygiene	H/o Diabetes	Duration of diabetes (years)	Immunosuppression	Redness	Pain	Swelling	Erythema	Crusting	Crusting (color)
76	673729	BK	60	F	OPD	4	Trunk	0.4	Y	N	-	N	Y	Y	Y	Y	N	-
77	845376	RV	30	F	OPD	4	Trunk	1	N	N	-	N	Y	Y	Y	N	N	-
78	364737	S	32	F	OPD	7	Trunk	2	N	N	-	N	Y	Y	Y	N	N	-
79	231407	GS	74	M	OPD	4	Trunk	1	N	N	-	N	Y	Y	Y	N	N	-
80	132036	DM	80	M	IPD	10	Lower limb	1	N	Y	10	Y	Y	Y	N	N	Y	dirty brown
81	848220	PS	66	M	OPD	14	Lower limb	2	N	Y	17	N	Y	Y	N	N	Y	dirty brown
82	866347	GK	60	F	OPD	4	Lower limb	1	N	Y	1	N	Y	Y	Y	N	Y	-
83	842293	R	46	M	OPD	2	Trunk	2	Y	N	-	N	Y	Y	Y	Y	N	-
84	888971	PS	45	M	IPD	9	Lower limb	1	N	N	-	N	Y	Y	N	N	N	dirty brown
85	893020	SS	42	M	OPD	6	Lower limb	3	N	Y	7	N	Y	Y	Y	N	Y	-
86	362688	GS	70	M	OPD	6	Head and neck	0.1	N	N	-	N	N	Y	N	N	N	-
87	874412	NK	38	M	OPD	7	Lower limb	3	N	N	-	N	Y	Y	Y	N	N	-
88	61417	MK	29	M	OPD	3	Head and neck	0.1	N	N	-	N	Y	N	N	N	N	-
89	888482	JS	62	M	OPD	4	Trunk	1	Y	N	-	N	Y	N	N	Y	N	dirty brown
90	96348	JD	64	M	OPD	10	Lower limb	2	N	N	-	N	Y	Y	Y	N	N	dirty brown
91	878772	PK	24	F	OPD	3	Trunk	0.3	Y	N	-	N	Y	Y	N	Y	N	-
92	858871	SK	54	M	OPD	15	Lower limb	3	N	N	-	N	Y	Y	Y	N	N	-
93	764593	SK	70	F	IPD	5	Trunk	4	N	Y	28	N	Y	Y	Y	N	Y	-
94	111779	S	43	F	OPD	6	Trunk	2	N	N	-	N	Y	Y	Y	N	N	-
95	295035	MK	52	F	IPD	5	Lower limb	2	N	N	-	N	Y	Y	N	N	N	-
96	420517	HK	33	F	OPD	5	Lower limb	0.2	N	N	-	N	Y	N	N	N	N	-
97	496368	A	52	M	IPD	3	Lower limb	1	N	N	-	N	Y	N	N	N	N	-
98	859654	RK	33	F	OPD	2	Trunk	1	N	N	-	N	Y	N	N	N	N	-
99	869896	A	28	F	OPD	3	Lower limb	0.2	N	N	-	N	N	N	N	N	N	-
100	864746	S	35	M	OPD	3	Trunk	0.1	N	N	-	N	Y	N	N	N	N	-

S.No.	MRD No.	s		Type of pyoderma	Clinical Diagnosis	Gram's staining	Identification		MRSA				
		Oozing	Oozing (color)				organism	organism		Cotrimoxazole	Amikacin	Cefoxitin	Ciprofloxacin
76	673729	N	-	Sec	Infected scabies	gram positive cocci in cluster,few pus cells	S.aureus	-	-	S	S	S	S
77	845376	N	-	P	Abscess	Many pus cells	no growth	-	-	-	-	-	-
78	364737	Y	Reddish-yellow	P	Abscess	gram positive cocci in cluster,few pus cells	S.aureus	-	-	S	S	S	S
79	231407	N	-	P	Furuncle	gram positive cocci in cluster,few pus cells	S.aureus	-	-	S	S	S	S
80	132036	N	-	P	Ecthyma	Many gram negative bacilli,many pus cells	P.aeruginosa	-	-	-	R	-	R
81	848220	N	-	P	Ecthyma	gram negative bacilli,many pus cells	E.coli	-	-	R	R	-	R
82	866347	N	-	P	Furuncle	gram positive cocci in cluster,few pus cells	S.aureus	-	-	R	S	S	R
83	842293	N	-	Sec	Infected tinea	gram positive cocci in cluster,few pus cells	S.aureus	-	-	S	S	S	R
84	888971	N	-	P	Ecthyma	gram negative bacilli,many pus cells	E.coli	-	-	R	R	-	R
85	893020	N	-	P	Folliculitis	gram positive cocci in cluster,few pus cells	S.aureus	-	-	S	S	-	R
86	362688	N	-	P	Folliculitis	many pus cells	no growth	-	-	-	-	-	-
87	874412	N	-	P	Ecthyma	gram positive cocci in cluster,few pus cells	S.aureus	-	-	S	S	S	R
88	61417	N	-	P	Folliculitis	Many pus cells	no growth	-	-	-	-	-	-
89	888482	N	-	Sec	Infected scabies	Many pus cells	no growth	-	-	-	-	-	-
90	96348	N	-	P	Ecthyma	gram positive cocci in cluster,few pus cells	S.aureus	-	-	S	S	S	R
91	878772	N	-	Sec	Infected tinea	gram positive cocci in cluster,few pus cells	S.aureus	-	-	S	S	S	R
92	858871	N	-	P	Cellulitis	gram positive cocci in cluster,few pus cells	S.aureus	-	Y	S	S	R	S
93	764593	Y	yellowish	P	Carbuncle	Many gram negative bacilli,many pus cells	K.pneumoniae	P.aeruginosa	-	R	S	-	MS
94	111779	N	-	P	Abscess	gram negative bacilli,many pus cells	P.aeruginosa	-	-	-	S	-	S
95	295035	N	-	P	Abscess	short round gram negative bacilli,many pus cells	Acinetobacter baumanii	-	-	S	S	-	S
96	420517	N	-	P	DCPA	gram positive cocci in cluster,few pus cells	S.aureus	-	-	S	S	S	S
97	496368	N	-	P	Furuncle	gram negative bacilli,Many pus cells	Proteus mirabilis	-	-	S	S	-	R
98	859654	N	-	P	Furuncle	Many pus cells	no growth	-	-	-	-	-	-
99	869896	N	-	P	Folliculitis	gram positive cocci in cluster,few pus cells	S.aureus	-	-	S	S	S	R
100	864746	N	-	P	Folliculitis	gram positive cocci in cluster,few pus cells	S.aureus	-	-	S	S	S	R

S.No.	MRD No.	Antibiotic susceptibility													
		Clindamycin	Erythromycin	Gentamycin	Linezolid	Penicillin	Teicoplanin	Vancomycin	Cefotaxime	Amoxiclav	Cefoperazone+ Sulbactam	Piperacillin	Piperacillin+Tazobactam	Oxacillin	Ticarcillin+clavulanic acid
76	673729	S	S	S	S	S	S	S	-	-	-	-	-	-	-
77	845376	-	-	-	-	-	-	-	-	-	-	-	-	-	-
78	364737	S	S	S	S	S	S	S	-	-	-	-	-	-	-
79	231407	S	S	S	S	R	S	S	-	-	-	-	-	-	-
80	132036	-	-	R	-	-	-	-	-	-	R	-	R	-	R
81	848220	-	-	R	-	-	-	-	R	-	-	S	S	-	-
82	866347	S	S	S	S	R	S	S	-	-	-	-	-	-	-
83	842293	S	R	S	S	R	S	S	-	-	-	-	-	-	-
84	888971	-	-	R	-	-	-	-	R	-	-	S	S	-	-
85	893020	S	S	S	S	S	S	S	-	-	-	-	-	-	-
86	362688	-	-	-	-	-	-	-	-	-	-	-	-	-	-
87	874412	S	S	S	S	R	S	S	-	-	-	-	-	-	-
88	61417	-	-	-	-	-	-	-	-	-	-	-	-	-	-
89	888482	-	-	-	-	-	-	-	-	-	-	-	-	-	-
90	96348	S	R	S	S	R	S	S	-	-	-	-	-	-	-
91	878772	S	R	S	S	R	S	S	-	-	-	-	-	-	-
92	858871	S	S	S	R	S	S	S	-	-	-	-	-	-	-
93	764593	-	-	-	-	-	-	-	R	-	S	MS	S	-	-
94	111779	-	-	S	-	-	-	-	-	-	S	S	S	-	-
95	295035	-	S	-	-	-	-	-	R	-	S	R	S	-	-
96	420517	S	S	-	S	S	S	S	-	-	-	-	-	-	-
97	496368	-	-	S	-	-	-	-	-	-	S	-	S	-	S
98	859654	-	-	-	-	-	-	-	-	-	-	-	-	-	-
99	869896	S	S	S	-	R	S	S	-	-	-	-	-	-	-
100	864746	S	S	S	-	R	S	S	-	-	-	-	-	-	-

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The Principal,
DMC, Ludhiana.

Subject: **Approval of Plan of Thesis.**

This is to inform you that the Faculty of Medical Sciences in its meeting held on 29.05.2019 has approved the plan of thesis of **Aditi Bansal**, a student of your college for **MD Dermatology, Venerology & Leprosy** along with the names of Supervisor/Co-Supervisor as under:-

Topic Plan of Thesis

Comparison of clinico-bacteriological profile of pyoderma in children and adults

Supervisor
Dr. Sunil Gupta
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The student has to submit the four copies of thesis (along with PDF format) , to the Controller of Examinations, BFUHS, Faridkot, at least six months before the commencement of theory examination, if he/she is otherwise eligible to take the examination. He /She must directed to attach a photocopy of this letter with the forwarding letter while submitting thesis to the University along with the letter of approval of University for change of supervisor/Co-Supervisor, change of topic,(if any).

SPSingh
Dr. S P Singh MS(Oph)
Controller of Examinations

Note :

1. *If any discrepancy found regarding topic of thesis or Supervisor/Co-Supervisor, inform undersigned within one month after issue of this letter.*
2. *The student may submit the thesis as per the schedule already conveyed i.e for May/June examination 30th Nov of the preceding the year of examination and for Nov/Dec examinations 31st May of the year of examination.*

COMPARISON OF CLINICO-BACTERIOLOGICAL PROFILE OF PYODERMA IN CHILDREN AND ADULTS

THESIS PROPOSAL

FOR APPROVAL OF SUBJECT OF THESIS TO BE SUBMITTED

IN PARTIAL FULFILMENT OF THE REQUIREMENTS

FOR THE DEGREE OF

Doctor of Medicine

in

DERMATOLOGY, VENEREOLOGY AND LEPROLOGY

OF THE

BABA FARID UNIVERSITY OF HEALTH SCIENCES

FARIDKOT

August, 2018

Dr. Aditi Bansal

DEPARTMENT OF DERMATOLOGY, VENEREOLOGY & LEPROLOGY
DAYANAND MEDICAL COLLEGE & HOSPITAL, LUDHIANA
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ABSTRACT OF PLAN OF THESIS

Title	Comparison of clinico-bacteriological profile of pyoderma in children and adults.
For the degree of	M.D. Dermatology, Venereology & Leprology
Name of the candidate	Dr Aditi Bansal
Supervisor	Dr. Sunil Gupta, Prof. and Head, Department of Dermatology, DMCH, Ludhiana
Co-supervisor	Dr. Veenu Gupta, Professor, Department of Microbiology, DMCH, Ludhiana Dr. Sukhjot Kaur Goraya, Associate Professor, Department of Dermatology, DMCH, Ludhiana
Institution	Dayanand Medical College and Hospital, Ludhiana
University	Baba Farid University of Health Sciences, Faridkot

Introduction: Pyogenic infections are characterized by local and systemic inflammation, formation of pus, generally caused by one of the pyogenic bacteria. This study will be conducted to determine the clinical and bacteriological profile in patients of pyoderma.

Aim of study: To study the clinical manifestations and bacteriological profile of pyoderma in children and adults.

Materials and methods: The study will be conducted at Dayanand Medical College and Hospital, Ludhiana on patients of pyoderma. A total of 200 patients of pyoderma will be included in the study to find out the pattern of disease in each group, causative organism and their antibiotic susceptibility pattern.

Clinical significance: To decrease the antimicrobial resistance which is increasing now a days due to empirical therapy given in pyodermas.

Keywords: Pyoderma, Antibiotic susceptibility

(Signature)	(Signature)	(Signature)	(Signature)	(Signature)
Candidate	Co-supervisor	Co-supervisor	Supervisor	Principal

CERTIFICATE OF FACILITIES AVAILABLE

This is to certify that the facilities for work on the subject of thesis titled "**COMPARISON OF CLINICO-BACTERIOLOGICAL PROFILE OF PYODERMA IN CHILDREN AND ADULTS**" exist in the Department of Dermatology, Venereology & Leprosy at Dayanand Medical College and Hospital, LUDHIANA and will be provided to the candidate. We will see that the data being included in the thesis are genuine and is collected by the candidate herself under our supervision and guidance.

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APPROVAL PROFORMA
BY
RESEARCH & ETHICAL COMMITTEE
DAYANAND MEDICAL COLLEGE & HOSPITAL, LUDHIANA

Name of candidate	Dr. AditiBansal
Department	DERMATOLOGY, VENEREOLOGY AND LEPROLOGY
Topic of Thesis	Comparison of clinico-bacteriological profile of pyoderma in children and adults.
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Date of enrollment	MAY 9, 2018
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1. HOD Pharmacology	1. HOD Physiology
2. HOD Surgery	2. HOD Pathology
3. HOD Pathology	3. HOD Pharmacology
4 HOD Medicine	4. HOD SPM
5. Dean Academics	5. HOD Medicine
6. Vice Principal	6. HOD Surgery
	7. HOD Obst. & Gynae
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	10. Dean Academics
	11. Vice Principal
	12. Principal

Approved: Yes / No

Chairperson
Research Committee

Chairperson
Research Committee

Approved: Yes / No

Chairperson
Ethics Committee

Chairperson
Ethics Committee

BABA FARID UNIVERSITY OF HEALTH SCIENCES, FARIDKOT

PROFORMA FOR REGISTRATION OF SUBJECT FOR DISSERTATION

1	NAME OF THE CANDIDATE AND ADDRESS	Dr Aditi Bansal
2	NAME OF THE INSTITUTE	Dayanand Medical College & Hospital, Ludhiana
3	COURSE OF STUDY AND SUBJECT	M.D. Dermatology, Venereology And Leprology
4	DATE OF ADMISSION OF COURSE	09-05-2018
5	TITLE OF THE TOPIC	COMPARISON OF CLINICO-BACTERIOLOGICAL PROFILE OF PYODERMA IN CHILDREN AND ADULTS

6. BRIEF RESUME OF THE INTENDED WORK

6.1 NEED FOR THE STUDY

Cutaneous pyogenic infections have been reported worldwide as being one of the most common human diseases in clinical practice. Inspite of therapeutic advances in last decades, the prevalence of cutaneous pyogenic infections is still increasing.

The incidence is high in the Indian subcontinent because of warm and humid climate, poverty, malnutrition, overcrowding, poor hygiene and lack of medical facilities. Bacterial infections resemble several diseases in the field of Dermatology and Medicine, so it is necessary to diagnose the infection with some laboratory evidence before starting treatment with antibiotics.

Since bacterial infections can cause physical, psychological and occupational problems, the clinico-epidemiological data can be helpful in creating public awareness and development of preventive and treatment strategies. Also, the prevalance of these bacterial infections vary with time and geographical location. Many cases these days don't respond to antibiotics that were previously very effective. Indiscriminate use of topical as well as systemic antibiotics have contributed to this grave situation. The emergence of antibiotic resistance has significantly eroded the utility of established antibiotics and poses a serious threat to public health worldwide.

So, a correct knowledge of the etiological agents of cutaneous pyogenic infections is therefore important to initiate appropriate treatment and is also essential for epidemiological purposes.

Thus, the present study is undertaken to shed light on the clinical manifestations of cutaneous pyogenic infections, identifying the causative

organism and their antibiotic susceptibility patterns and to correlate clinical diagnosis with gram staining and pus culture and sensitivity among the patients of different age groups attending the outpatient and inpatient Department of Dermatology in Dayanand Medical College and Hospital, Ludhiana.

6.2 REVIEW OF LITERATURE

Skin, the largest organ in the human body, plays a crucial role in the sustenance of life through regulation of water and electrolyte balance, thermoregulation, and by acting as a barrier to external noxious agents including microorganisms, however. when the epithelial integrity of skin is disrupted, a wound results . This may be characterized by the classic signs of redness, pain, swelling, raised temperature and fever.[1]

Pyodermas are quite common in India and constitute a major portion of the cases in dermatology clinics. Pyodermas may be milder or severe and it forms are many with different etiological microbial and non -microbial agents, and it is influenced by several predisposing factors. Primary pyoderma is a pyogenic infection of non-diseased skin and its appendages.[2] It includes Impetigo, Folliculitis, Furunculosis, Carbuncle and Ecthyma. The secondary pyoderma is a pyogenic infection of previously diseased skin and it includes Infected Eczema, Infected Scabies, Infected Wounds and Trophic Ulcers.[3]

Pyogenic infections are characterized by local and systemic inflammation, formation of pus, generally caused by one of the pyogenic bacteria, which result in the aggregation of dead leukocytes as well as pyogenic bacteria commonly known as pus, "a white to yellow fluid". [4-6]

Pyogenic Infections may be endogenous or exogenous. A break or abrasion in the skin can provide an entryway for these surface bacteria into the body, and they stick very well to the moist edges of a cut.[4]

Wound infection can be caused by variety of organisms like bacteria. virus, fungi and protozoa and may co-exist as poly microbial communities especially

in wound margins and in chronic wounds [7] and in many cases there is a mixed infection with more than one bacterial species.[7]

The majority of pyodermas are caused by Gram positive cocci, particularly Staphylococcus aureus and Streptococcus pyogenes. Other organisms that may rarely cause pyodermas include coagulase negative *Staphylococci*, *Streptococcus faecalis*, *Streptococcus pneumonia*, Gram negative bacilli [8]–*Escherichia coli* and other *coliform*, *Proteus spp.*, *Providencia spp.*,*Klebsiella spp.*,*Morganella spp.*, *Enterobacter spp.*, *Cirobacter spp.*, anaerobic bacteria, *Pseudomonas spp.*, *Haemophilus influenzae and Bacillus cereus*.[9,10] Although most pyodermas are self-resolving, some may be complicated by glomerulonephritis, especially where *Streptococcus pyogenes* is the causative organism.[11]

Factors affecting infections are age, sex, genetic and racial factors, endocrine and metabolic factors, temperature and microenvironment, competing organisms and co-pathogens.

Even though the bacterial profile of pus samples in many studies remain the same, the antibiotic resistance pattern of these isolates has shown a lot of variations.[12,13] The inadvertent use of antibiotics leads to emergence of drug resistant pathogens, which in turn acts as a great challenge to the health services. Moreover, highly virulent strains and capacity to adapt quickly to changing environment worsens the situation and draws a matter of concern.[14]

The emergences of multi-drug resistant strains have resulted in prolonged illness, higher health care expenditures and higher risk of death due to infection. Antimicrobial resistance in addition hampers the control of infectious

diseases by reducing the effectiveness of treatment thus patients remain infectious for a long time increasing the risk of spreading resistant microorganisms to others.[15-16]

During the last few decades, multidrug-resistant Gram-negative bacterial strains such as *Acinetobacter baumannii, Escherichia coli, Klebsiella pneumoniae, Pseudomonas aeruginosa, and* Gram-positive *methicillin-resistant Staphylococcus aureus* (MRSA) were increasingly associated with pus infections under hospital settings due to extensive misprescription and inadequate dose regimen of antibiotics.[15,16]

For clinical diagnosis:

1. The detailed history of patient is essential regarding age, occupation, hobbies, living conditions with onset, duration and course of disease as well as intake of previous treatment and presence of any family history.
2. The clinical examination is done in well-lit room, with magnifying lens. The site of lesion, morphology of lesion, signs and symptoms are essential.

Laboratory diagnosis is done by:

1. Direct microscopic examination: It can be done by Gram's stain.
2. Culture: Swabs are collected from the lesion and are inoculated onto Blood Agar and Mac Conkey Agar for culture as per standard laboratory procedures. The organisms isolated are identified on the basis of colony morphology, culture characteristics and biochemical tests such as catalase, coagulase, oxidase, indole, methyl red, voges-proskauer, citrate, urease and phenyl pyruvic acid test.
3. Antibiotic sensitivity test: It is done by Kirby Bauer Disc diffusion method using Muller Hinton agar as per CLSI guidelines.[16]

4. Molecular analysis have recently been applied.

In a study of 387samples, Gram positive and Gram negative organisms isolated were 47.36% and 51.97% respectively. The maximum (93.3%) isolates were resistant to penicillin, followed by Cotrimoxazole (67.3%) and Erythromycin (50%).

Resistance to Linezolid was observed in 2.9% cases while vancomycin and Teicoplanin showed 1.9 % and 6.7% resistance. it was observed that 26.9 % isolates were MRSA.[20]

In a study of 149 samples, predominant isolate was *S. aureus* followed by *E.coli, Pseudomonas, Klebsiella, Proteus.*

S.aureus has maximum Amoxicillin(89.47%) resistance followed by Cotrimoxazole (69.23%), Ciprofloxacin(29.41%), Ceftriaxone (30%).Gram negative bacilli had highest resistance to Amoxicillin(100%),Cefipime(50-100%),Cefotaxime(50-100%),Ceftriaxone(33.33-100%) [21]

Since bacterial infections can cause physical, psychological and occupational problems, so a correct knowledge of the etiological agents of cutaneous bacterial infections is therefore important to initiate appropriate treatment, and also is essential for epidemiological and treatment purposes.

6.3 AIMS AND OBJECTIVES OF THE STUDY

1. To study the clinical manifestations of pyoderma in children and adults.
2. To study their etiological agents and correlate clinical and bacteriological findings.

7. MATERIALS AND METHODS

7.1 Source of Data

Patients attending outpatient and inpatient Department of Dermatology of Dayanand Medical College and Hospital, Ludhiana, will constitute the study material and the source of data.

7.2METHOD OF COLLECTION OF DATA

INCLUSION CRITERIA:

A total of two hundred clinically suspected cases of pyoderma attending outpatient and inpatient Department of Dermatology of Dayanand Medical College and Hospital, Ludhiana, will be included in the study.

EXCLUSION CRITERIA:

- Patients not willing to participate
- Patients already on Antibiotic therapy
- Patients who took any antibiotic in last 7 days

METHOD:

A total of two hundred clinically suspected cases of pyodermas will be included in this study. These two hundred patients will be divided into two groups i.e. children upto 18 years and adult more than 18 years. Detailed history of the patients including duration of disease, history of previous treatment taken will be taken. Specimen like pus will be collected and processed in the Department of Microbiology. Gram staining and Culture and sensitivity of all the samples will be done.

The following data will be collected:

1. Patient's details including name, age, sex, CR No., history (past, personal, family, treatment)

2. Data of collection of samples

3.Local examination findings

4.Provisional clinical diagnosis

5.Gram staining findings

6.Pus Culture and senstivity report

Number of cases

Two Hundred

Procedure of sample collection and transportation

Pus samples will be collected using sterile cotton swabs placed in the screw capped container and pus aspirates will be collected by using sterile disposable syringes.

Specimen transportation

Pus/wound swab samples will be collected and transported in a clean, sterile container for processing in Microbiology Department.

Processing of specimens

Pus/Wound swab samples will be processed in the Department of Microbiology as follows:

1. DIRECT MICROSCOPIC EXAMINATION: It will be done by Gram's stain. Gram positive organisms stain violet, while Gram negative organisms stain red against a light pink background.
2. CULTURE: Swabs will be collected from the lesion and will be inoculated onto Blood Agar and Mac Conkey Agar for culture as per standard laboratory procedures. The organisms isolated will be identified on the basis of colony morphology and various biochemical tests such as

catalase,coagulase,indole,methylred,oxidase,citrate,urease,phenyl pyruvic acid, voges-proskauer test.

3. ANTIBIOTIC SENSITIVITY TEST: It will be done by Kirby Bauer Disc diffusion method using Muller Hinton agar as per CLSI guidelines.[19]

The antimicrobial agents, which will be tested: Ampicillin (10µg), amoxicillin / clavulinic acid (20/10µg), gentamycin (10µg), cotrimoxazole (25µg) Ciprofloxacin (5µg), erythromycin (15µg), cephalexin (30µg), linezolid (30µg), vancomycin (30µg), Teicoplanin (30 µg) for Gram positive organisms.

For Gram negative organisms, amikacin (30µg), gentamycin (10µg), ciprofloxacin (5µg), piperacillin (100µg), piperacillin + tazobactum (100/10µg), Cefotaxime (30µg).

The diameter of zone of inhibition will be measured and interpreted as susceptible, intermediate or resistant.

For detection of MRSA cefoxitin disc diffusion technique will be used as per CLSI guidelines.

The clinico-bacteriological profile of cutaneous bacterial infections will be determined and the data obtained will be put to relevant descriptive analyses.

Outcome

It will be evaluated in view of –

1.Pattern of disease in each group

2.Type of bacteria found

3.Antibiotic sensitivity to isolated bacteria

Statistical Analysis

Data will be described in terms of range; mean ± standard deviation (± SD), median, frequencies (number of cases) and relative frequencies

(percentages) as appropriate. For comparing categorical data, Chi square ($\chi 2$) test will be performed and exact test will be used when the expected frequency is less than 5. A probability value (p value)less than 0.05 will be considered statistically significant. All statistical calculations will be done using SPSS (Statistical Package for the Social Science) SPSS 21 version statistical program for Microsoft Windows.

7.3 DOES THE STUDY REQUIRE ANY INVESTIGATIONS OR INTERVENTIONS TO BE CONDUCTED ON PATIENTS OR OTHER HUMANS OR ANIMALS? IF SO, PLEASE DESCRIBE BRIEFLY.

The following investigations will be advised to patients for diagnosing pyoderma.

1. Gram's staining
2. Pus culture and sensitivity

The study does not require any animal experimentation and/or human intervention.

7.4 HAS ETHICAL CLEARANCE BEEN OBTAINED FROM YOUR INSTITUTION IN CASE OF 7.3?

Attached herewith.

8. LIST OF REFERENCES

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PROFORMA

DATE-

Name :

S.NO.-

Age: Sex: MRD NO.-

Father/Husband's name:

Occupation:

Address:

Contact No:

Date of collection of sample:

Specimen:

Clinical presentation

-Primary/Secondary Pyoderma:

-Duration of disease:

Morphology of lesion- Site Size erythema
Crust(Color) Oozing(Color)

Symptoms- Redness Pain Swelling Other

Signature of Candidate **Signature of Supervisor**

Aggravating factors- Poor hygiene Diabetes

Immunosuppression · Others

Nutrition and Built

History

-Past history

-Personal history

-Family history

-Treatment history

Clinical diagnosis

Microscopy

-Gram's staining

-Identification

Pus Culture-Isolated organism

Antibiotic susceptibility

Signature of Candidate **Signature of Supervisor**

INFORMED PATIENT CONSENT

Date: _____

I.....D/o, W/o, S/o.....

.....am willing to participate in the study **"COMPARISON OF CLINICO-BACTERIOLOGICAL PROFILE OF PYODERMA IN CHILDREN AND ADULTS"** The procedure with its complications has been explained to me in local vernacular language. I have fully understood that no special tests and extra expenditure in any form, merely for the purpose of thesis is being carried out. I am free to opt out the study any time without assigning any reason.

....
Signature of the Witness

Name:

.....
Signature of the patient

Signature of Supervisor

Signature of Candidate

मरीज़ अनुमति फार्म

तिथि _____

मैं _____ पत्नी/पुत्र/पुत्री _____ _____

निवासी _____

इस शोधकार्य "**"COMPARISON OF CLINICO-BACTERIOLOGICAL PROFILE OF PYODERMA IN CHILDREN AND ADULTS"** में भाग लेने के लिए सहमत हूँ। मैं इस शोधकार्य में भाग लेने के लिए अपनी स्वेच्छा से तैयार हूँ। मैं पुष्टि करता/करती हूँ की यह स्वीकृति देने के लिए मुझे कोई आर्थिक प्रलोभन नहीं दिया गया और न ही दबाव डाला गया है। मुझे इस शोधकार्य का उद्देश्य स्पष्ट कर दिया गया है और मुझे यह विश्वास है की यह विज्ञान और मानवता के लाभ के लिए है।

मैं इस शोधकार्य में भाग लेने के सम्बन्ध में किसी भी समय अपनी अस्वीकृति व्यक्त कर सकता/सकती हूँ और मेरे अस्वीकृति व्यक्त करने का मेरे इलाज पर कोई प्रभाव नहीं पड़ेगा।

_____ _____

गवाह के हस्ताक्षर रोगी के हस्ताक्षर/अंगूठे का निशान

नाम: नाम:

_____

अन्वेषक के हस्ताक्षर

_____

विद्यार्थी के हस्ताक्षर

ਮਰੀਜ਼ ਰਜ਼ਾਮੰਦੀ ਫਾਰਮ

ਮਿਤੀ _____

ਮੈ_____ _____ਪਤਨੀ/ਪੁੱਤਰ/ਧੁੱਤਰੀ_____

ਵਾਸੀ _____

"COMPARISON OF CLINICO-BACTERIOLOGICAL PROFILE AND ANTIBIOTIC SUSCEPTIBILITY OF PYODERMA IN CHILDREN AND ADULTS" ਤੇ ਚਲ ਰਹੀ ਖੋਜ ਵਿਚ ਹਿੱਸਾ ਲੈਣ ਲਈ ਰਜ਼ਾਮੰਦ ਹਾਂ। ਇਸ ਖੋਜ ਕੰਮ ਵਿਚ ਹਿੱਸਾ ਲੈਣ ਲਈ ਮੈ ਆਪਣੀ ਮਰਜ਼ੀ ਨਾਲ ਜ਼ਿਆਦਾ ਹਾਂ। ਮੈ ਇਹ ਦਸਣਾ ਚਾਹੁੰਦਾ ਹਾਂ/ ਚਾਹੁੰਦੀ ਹਾ ਕੇ ਇਹ ਰਜ਼ਾਮੰਦੀ ਦੇਣ ਵਾਸਤੇ ਮੈਨੂ ਕਿਸੇ ਵੀ ਤਰਾਂ ਦਾ ਲੋਭ-ਲਾਲਚ ਨਹੀ ਦਿੱਤਾ ਗਿਆ ਅਤੇ ਨਾ ਹੀ ਕੋਈ ਦਬਾ ਪਾਇਆ ਗਿਆ ਹੈ। ਮੈਨੂ ਇਸ ਖੋਜ ਦਾ ਮਨੋਰਥ ਸਮਝਾ ਦਿੱਤਾ ਗਿਆ ਹੈ ਅਤੇ ਮੈਨੂ ਇਹ ਭਰੋਸਾ ਦਵਾਇਆ ਗਿਆ ਹੈ ਕੇ ਇਹ ਖੋਜ ਵਿਗਿਆਨ ਅਤੇ ਮਾਨਵਤਾ ਦੀ ਭਲਾਈ ਲਈ ਹੈ।

ਮੈ ਇਸ ਖੋਜ ਕੰਮ ਵਿਚ ਹਿੱਸਾ ਲੈਣ ਬਾਰੇ ਆਪਣੀ ਰਜ਼ਾਮੰਦੀ ਕਿਸੇ ਵੀ ਸਮੇ ਵਾਪਸ ਲੈ ਸਕਦਾ ਹਾਂ/ ਸਕਦੀ ਹਾ ਅਤੇ ਮੇਰੀ ਰਜ਼ਾਮੰਦੀ ਵਾਪਸ ਲੈਣ ਦਾ ਮੇਰੇ ਇਲਾਜ ਤੇ ਕੋਈ ਅਸਰ ਨਹੀ ਹੋਵੇਗਾ।

_____ _____

ਗਵਾਹ ਦੇ ਹਸਤਾਖਰ ਮਰੀਜ਼ ਦੇ ਹਸਤਾਖਰ

ਮਿਤੀ_____ ਮਿਤੀ_____

ਪਤਾ _____ ਪਤਾ _____

_____

ਨਿਰੀਖਕ ਦੇ ਹਸਤਾਖਰ

_____

ਸਿਖਿਆਰਥੀ ਦੇ ਹਸਤਾਖਰ